

Interactions of Physical Training and Heat Acclimation

The Thermophysiology of Exercising in a Hot Climate

Yukitoshi Aoyagi,¹ Tom M. McLellan^{1,2} and Roy J. Shephard^{1,4}

1 Graduate Department of Community Health, University of Toronto, Toronto, Ontario, Canada

2 Defence and Civil Institute of Environmental Medicine, North York, Ontario, Canada

3 School of Physical and Health Education, University of Toronto, Toronto, Ontario, Canada

4 Health Studies Programme, Brock University, St Catharines, Ontario, Canada

Contents

Summary	174
1. Terminology and Units	174
2. Energy Exchange, Energy Balance, and Body Temperature	175
2.1 Metabolic Heat Production (M)	175
2.2 External Power (W)	180
2.3 Convective (C_{res}) and Evaporative (E_{res}) Heat Exchanges through the Respiratory Tract	181
2.4 Radiant (R), Convective (C) and Conductive (K) Heat Exchanges at the Skin Surface	181
2.5 Evaporative Heat Loss from the Skin Surface (E_{sk})	188
3. Conventional Methods of Estimating Body Heat Storage	189
3.1 Specific Heat of the Body ($C_{p,b}$)	190
3.2 Weighting Factors for Local Skin Temperatures	191
3.3 Relative Weightings of Core Temperature (T_c) and Mean Skin Temperature (T_{sk})	192
4. Tolerance of Exercise and Heat Stress	193
4.1 Constraints Imposed by Impermeable and Protective Clothing	193
4.2 Modifications of the Response to Heat-Exercise Stress Induced by Physical Training and by Heat Acclimation	194
4.3 Criteria of Heat-Exercise Tolerance	195
4.4 Modifications of Heat-Exercise Tolerance by Physical Training and by Heat Acclimation	195
4.5 Interactions of Physical Training and Heat Acclimation	196
5. Factors Affecting Physical Training and Heat Acclimation	197
5.1 Hydration	197
5.2 Blood Doping: Artificial Expansion of Plasma Volume	199
5.3 Carbohydrate Replacement	199
5.4 Electrolyte Replacement	199
5.5 Menstrual Cycle	200
5.6 Circadian Rhythms	200
5.7 Sleep Deprivation	201
5.8 Gender and Age	201
6. Practical Recommendations to Coaches and Sports Physicians	201
6.1 Planning of Physical Training Programmes	201
6.2 Heat Acclimation and Its Planning	202
7. Conclusions	203

Summary

Physical training and heat acclimation are both commonly adopted tactics to improve performance and/or tolerance times when individuals must compete or work in the heat. Potential benefits include: (i) improved aerobic fitness and thus a greater cardiovascular reserve (probably seen mainly after training); (ii) a lower resting body temperature that allows greater heat storage (probably seen mainly after acclimation); (iii) a decreased energy cost of a given intensity of exercise (seen after acclimation and also as the learning component of training); (iv) an enhanced sweating response at a given percentage of maximal effort (probably developed by both treatments); (v) a slower increase in body temperature owing to (iii) and/or (iv) [seen after both treatments]; (vi) a reduced cardiovascular stress because of changes in the autonomic nervous system (probably realised mainly by training), expansion of blood volume (seen after both treatments) and/or a decreased peripheral pooling of blood (probably found after both treatments); and (vii) improved subjective tolerance reflecting a decrease in the relative intensity of a given activity (probably seen mainly after training), a reduction in the physiological strain (found after both treatments) and/or habituation to heat-exercise stress (probably developed by both treatments). Factors affecting improvements in physiological and psychological responses to a given set of conditions include: (i) the individual's initial fitness and acclimatisation to heat; (ii) age, gender, hydration, sleep deprivation, circadian rhythms and in women the menstrual cycle; (iii) use of ergogenic aids such as fluid ingestion, carbohydrate and/or electrolyte replacement and blood doping; (iv) event or test conditions such as the mode of exercise, the severity of environmental heat stress and the type of clothing worn; and (v) treatment conditions such as the intensity, duration and frequency of exercise and/or heat exposure, the length of any rest intervals and cumulative depletion of body water and minerals.

Exposure to thermal stress is a major threat to both the performance and the health of athletes who must compete in hot climates, particularly if the competitor's home base lies in a temperate region of the globe. This article provides an overview of the body's mechanisms of achieving heat balance, the influence of protective clothing and of physical training and heat acclimation on responses to exercise in the heat, and possible interactions between the 2 types of preparation for hot conditions. It covers mainly human research published between 1970 and 1996. For reviews of earlier literature, see other publications.^[1-11]

Some published research (for instance, Aoyagi et al.^[12-17]) refers to individuals who are wearing protective clothing. Although such studies were conceived in an occupational context, they have relevance to athletes who wear either impermeable

training suits or protective clothing for events such as football games.

1. Terminology and Units

We have followed the practice of Shephard,^[18] and made the distinction between acclimation, acclimatisation and adaptation. Acclimation was originally conceived as the process of becoming habituated to a new environment, although the term is now used to describe the totality of physiological adjustments to a novel environment that develop under controlled laboratory conditions. Acclimatisation is the term for functional compensations made over several weeks of exposure to the totality of a challenging new environment. Adaptation reflects more long term adjustments, including possible genetic influences, which favour life in a specific adverse environment.

All units for heat exchange and heat balance have, for the sake of consistency, been expressed in $\text{kJ} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$ rather than $\text{W} \cdot \text{m}^{-2}$; $1 \text{ kJ} \cdot \text{m}^{-2} \cdot \text{h}^{-1} = 0.278 \text{ W} \cdot \text{m}^{-2}$.

2. Energy Exchange, Energy Balance, and Body Temperature

The recreational exerciser and the competitive athlete normally compensate well for moderate climatic and metabolic heat stress by a combination of thermoregulatory and cardiovascular homeostatic mechanisms. Heat gains and losses reach a new level of equilibrium according to the First Law of Thermodynamics, as expressed in the classical heat balance equation:

$$S = M - (\pm W) \pm C_{\text{res}} - E_{\text{res}} \pm R \pm C \pm K - E_{\text{sk}} \quad (\text{Eq. 1})$$

where S is the quantity of heat stored in the body; M is the internal heat produced by metabolism; W is the external work performed (where $+$ is positive work, representing energy leaving the system, and $-$ is negative work); C_{res} is respiratory convective heat exchange; E_{res} is respiratory evaporative heat loss; R , C , and K are the quantities of heat exchanged by radiation, convection, and conduction through the skin surface, respectively; and E_{sk} is the heat lost by evaporation from the skin surface.

However, homeostatic mechanisms can be overwhelmed under severe conditions, including: (i) combinations of a high radiant heat load, a high air or water temperature and a high air or water velocity; (ii) combinations of more moderate climatic heat stress with a sustained high metabolic rate; (iii) a high relative humidity of ambient air; and (iv) clothing with limited vapour permeability that is worn for purposes of training or protection from physical injury. Specific examples include: (i) high values of R encountered when running outdoors in bright sunshine for 30 minutes or longer, high values of C (and C_{res}) encountered when exercising in desert heat and high values of C and K encountered when swimming in very warm water; (ii) high values of M found at a given value of W (for instance, when a poorly trained individual

performs an activity with a low mechanical efficiency); and (iii) low values of E_{sk} (and E_{res}), for instance, when exercising in hot and humid environments such as the southern US.

Behavioural modifications, physical training, and heat acclimation are tactics commonly adopted by both the sports physician and the industrial physiologist to meet the demands imposed by heat stress. Thus, a lowering of M through task learning and a resulting increase of mechanical efficiency, and a lowering of R by exercising when the sun has set and/or a training- or acclimation-induced enhancement of E_{sk} offer possibilities for the exerciser to slow the increase of body heat content and thus the rise in body temperature during a given bout of physical activity. Habituation further reduces the psychological distress associated with performance of a given activity in a particular climate.

2.1 Metabolic Heat Production (M)

2.1.1 Methods of Estimating M

In a few specialised laboratories, metabolic heat production (M) can be determined by direct calorimetry, but most exercise scientists estimate M much more simply, using mean values for oxygen consumption ($\dot{V}\text{O}_2$) in L/min at standard temperature and pressure, dry (STPD) and the respiratory quotient (RQ):^[2]

$$M = [(0.23 \cdot RQ + 0.77) \cdot 21.13] \cdot (60 \cdot \dot{V}\text{O}_2) \cdot A_D^{-1} \quad (\text{Eq. 2})$$

The RQ is calculated on the assumption that the exchange of O_2 and carbon dioxide measured at the lungs reflects the actual gas exchange from cellular metabolism:

$$RQ = \dot{V}\text{CO}_2 \cdot \dot{V}\text{O}_2^{-1} \quad (\text{Eq. 3})$$

where $\dot{V}\text{CO}_2$ is the rate of carbon dioxide production (in L/min STPD), and A_D is the body surface area (in m^2), estimated from body mass (Wt in kg) and height (Ht in m):^[19]

$$A_D = 0.202 \cdot Wt^{0.425} \cdot Ht^{0.725} \quad (\text{Eq. 4})$$

The term $(0.23 \cdot RQ + 0.77) \cdot 21.13$ in Eq. 2 is the estimated nonprotein energy equivalent of the O_2 consumed (in kJ/L).

Note that in the context of vigorous exercise, the estimate of M from gas exchange data can be distorted by: (i) an increase in the proportional contribution of protein to total metabolism as glycogen reserves are depleted; and (ii) a lack of equivalence between the cellular RQ and the respiratory gas exchange ratio (R) during and following brief, non-steady-state bursts of physical activity. R may diverge from RQ because: (i) the lactic acid generated during anaerobic exercise is compensated by an elimination of CO_2 (RQ and M are then overestimated); (ii) CO_2 is subsequently retained to replenish bicarbonate buffers (RQ and M are then underestimated); or (iii) an excess of CO_2 is eliminated (for example, anxiety hyperventilation prior to or during competition^[20] or thermal hyperventilation because body temperature is increased^[13,14]).

To avoid problems from an over- or underestimation of RQ , M may be estimated by an alternative equation, based on the fraction of O_2 utilised [the fraction of O_2 in inspired air (F_{IO_2}), a value of 0.2093 for normal air] minus the fraction of O_2 in expired air (F_{EO_2}) and the expired ventilation rate ($\dot{V}_E$) in L/min STPD:^[6]

$$M = 20.6 \cdot \{60 \cdot [\dot{V}_E \cdot (F_{IO_2} - F_{EO_2})] \cdot A_D^{-1} \quad (\text{Eq. 5})$$

The constant 20.6 is the commonly assumed energy equivalent of the O_2 consumed (in kJ/L). It supposes a constant nonprotein RQ of about 0.89, possibly somewhat low for the first hour of vigorous exercise. The term $\dot{V}_E \cdot (F_{IO_2} - F_{EO_2})$ is only equal to $\dot{V}O_2$ if the inspired respiratory minute volume ($\dot{V}_I$) is equal to $\dot{V}_E$ [and the nitrogen (N_2) concentration in inspired air (F_{IN_2}) is equal to that in expired air (F_{EN_2})]. During light activity, $\dot{V}_I > \dot{V}_E$ (and $F_{IN_2} < F_{EN_2}$), but during the first hour or more of vigorous exercise, the RQ approximates unity.

2.1.2 Modifications of M by Physical Training and by Heat Acclimation

If an activity is not fully learned, practice and/or training can lead to a progressive decrease in the

O_2 cost of the activity.^[21] The overall effect is no more than 5 to 10% for a simple task such as bench stepping or treadmill running, but it can be very large for a novice who begins to learn a skilled athletic activity such as swimming. Some investigators have reported that heat acclimation is also associated with a decreased O_2 cost of submaximal activities such as bench stepping,^[22,23] treadmill walking^[13,14,24-26] and cycle pedalling,^[27] but other authors have disputed this claim.^[28-30] Heat acclimation might reduce resting metabolism and increase heat dissipation, so that if prolonged exercise were to be performed under difficult conditions, the individual would be further from heat exhaustion (a situation where falling cerebral blood flow would lead to poorly coordinated and mechanically inefficient movement). The extent to which heat acclimation can reduce the metabolic cost of a given activity depends on: (i) the exercise mode; (ii) the extent to which the task is already learned; (iii) the test environment; (iv) the type of clothing worn; and (v) the length of acclimation. In general, the largest decrease of energy cost is seen when a skilled task must be pursued to near exhaustion and a long period of heat acclimation has been allowed.^[13,14,26]

Most of the empirical data relate to laboratory exercise. Decreases in metabolic cost after heat acclimation have been greater for rhythmic bench-stepping exercise (average reduction 13%, range 7 to 17%)^[22,23] than for either treadmill-walking (average reduction 4%, range 0 to 12%)^[13,14,24-26] or cycle-ergometer exercise (average reduction 1%, range 0 to 2%).^[27-30] Presumably, walking (albeit on an inclined treadmill) and cycling (albeit on a fixed cycle ergometer) are more familiar activities than rapid bench climbing. Sawka et al.^[26] observed that the percentage decrease of metabolic rate after heat acclimation was greater during treadmill walking in a cool (-5%) than in a hot (-3%) test environment. Aoyagi et al.^[13] further found that the reduction in O_2 cost after heat acclimation was more marked when wearing normal indoor clothing (-5%) than when wearing protective clothing (at most -2%). In the latter situation, the

microenvironment under the clothing is hotter and wetter,^[31,32] and enhancement of normal heat regulating mechanisms can do little to increase heat loss. The decrease in metabolism is larger for a longer period of heat acclimation (6 vs 12 days: -4% vs -11% for normal clothing, -3% vs -5% for protective clothing),^[14] although as with more intensive exercise,^[13] benefit from acclimation is greater with normal than with protective clothing. Such findings support the view that at least a part of the decrease in metabolism depends on evaporation of sweat.

2.1.3 Mechanisms of Reduced Energy Expenditure

There are at least 8 potential mechanisms to explain the reduced energy cost of exercise after heat acclimation:^[11,13,26] (i) learning of and habituation to the test exercise; (ii) improved coordination in an individual who is less distressed by the cumulative effects of hot environmental conditions; (iii) an acclimation-induced decrease of thyroid hormone secretion; (iv) a reduction of cardio-pulmonary work rate at any given intensity of exercise; (v) a Q_{10} effect; (vi) a decreased percentage contribution of anaerobic metabolism to the performance of vigorous exercise; (vii) greater muscular efficiency; and (viii) a greater recruitment of slow-twitch motor units. Depending on the initial status of the exerciser, several of these mechanisms (i, ii, iv, vi, vii, and viii) might also be developed over a period of physical training.

Rhythmic bench-stepping exercise requires moderate coordination, giving some scope for task learning. An exhausted individual also develops body sway and excessive limb movements, increasing energy expenditure. A similar picture is shown by a distance runner on a hot day. The mechanical efficiency of stepping can thus be increased by a combination of task learning with physical training and/or heat acclimation that reduces the likelihood of exhaustion. However, learning-, training- and acclimation-induced changes in mechanical efficiency are less likely for moderate periods of treadmill walking and cycle pedalling; these activities are familiar and require no

new skills [thus discounting mechanisms (i) and possibly (ii)]. Such considerations could explain why acclimation-induced decreases in energy cost have been larger for bench-stepping^[22,23] than for either treadmill-walking^[13,14,24-26] or cycle-ergometer exercise.^[27-30] Nevertheless, if the exerciser is wearing heavy, bulky protective clothing with a limited vapour permeability, the mechanical efficiency of treadmill walking or performance on the sports field could still decrease as exhaustion is approached,^[33,34] with potential for enhanced mechanical efficiency or performance in response to better training or acclimation [validating mechanism (ii)].

The thyroid gland produces 2 iodoamino acid hormones, 3,5,3'-tri-iodothyronine (T_3) and 3,5,3',5'-tetraiodothyronine (T_4 , thyroxine), that are important in stimulating oxidative metabolism and generating additional body heat in cold environments.^[35,36] An increase in T_3 and/or T_4 secretion raises the resting metabolic rate by uncoupling carbohydrate and lipid metabolism from phosphorylation, at least in the liver.^[37] Both laboratory and seasonal exposures to moderate heat can depress thyroid gland activity, with decrements in T_4 and/or T_3 levels and increments in reverse T_3 , the noncalorigenic metabolite of T_3 .^[35,38] Thus, a part of the lowering of metabolic rate with acclimation may be secondary to a depression of thyroid function [validating mechanism (iii)].

The decrease of exercise heart rate (HR), blood pressure, and pulmonary ventilation after heat acclimation should reduce the O_2 cost of cardiac and respiratory work. Physical training also decreases HR, blood pressure, and ventilatory responses to a given intensity of exercise. However, the resultant change in metabolism is small.^[13,26,39] Even during peak effort, cardiac and respiratory work together account for only about 5% of total metabolism,^[40] and any small decrease in the cardiorespiratory cost of exercise is usually submerged in the overall estimate of metabolism^[26] [thus discounting mechanism (iv)].

Over a limited range of temperatures, the velocity of enzyme-catalysed reactions increases 2- to 3-fold for a 10°C rise in tissue temperature

(the Q_{10} effect).^[41,42] Thus, even a small training- or acclimation-induced reduction of body temperature may decrease energy expenditures both in working muscles and elsewhere in the body^[43,44] [accepting mechanism (v)]. The Q_{10} effect is larger after heat acclimation than after physical training, because heat acclimation produces a greater reduction of body temperature in the exerciser; it lowers the thermoregulatory 'set point',^[3,11] reducing the resting body temperature,^[13,14,45] and decreasing thresholds for sweating^[46] and vasodilation^[47] during physical activity. The O_2 cost of exercise remains reduced over an entire 120- to 150-minute session of treadmill walking,^[13,14] and to accept a Q_{10} explanation, it is necessary to postulate that the body temperature of a heat-acclimated individual also remains lower than that of a nonacclimated person throughout an exercise bout.

Many^[27,28,48] but not all^[49-51] investigators have noted that dependence on anaerobic metabolism is greater in the heat than in cooler conditions. Contributing factors include a redistribution of blood flow from the exercising muscles to the skin and a greater recruitment of fast-twitch motor units. Heat acclimation increases mitochondrial capacity for free fatty acid oxidation,^[29] with a concomitant decrease in the rate of glycogen utilisation,^[28,29,52] so that muscle lactate accumulation is reduced after heat acclimation.^[27] Glycogen sparing is also observed after endurance training.^[53-55] The resultant lowering of RQ ^[27,29,54] in itself would make only a minor contribution to our estimate of the change in metabolism (Eq. 2). The metabolism of carbohydrate is also thermodynamically less efficient than the metabolism of fat if lactate is first produced, and is then transported elsewhere for oxidation or gluconeogenesis.^[56] However, mechanism (vi) is at most a second-order factor.

The chemical efficiency of the muscle reflects the product of the ratio of mechanical work output to the free energy of hydrolysis of adenosine triphosphate (ATP) [contractile-coupling efficiency] and the ratio of the free energy conserved as ATP to the free energy of oxidised foodstuff (phos-

phorylative-coupling efficiency).^[43,57,58] Slow-twitch muscle fibres have a higher contractile-coupling efficiency than fast-twitch muscle fibres.^[58,59] The phosphorylation efficiency of isolated skeletal muscle averages about 60%^[58] at temperatures between 25 and 40°C.^[43] Phosphorylation efficiency begins to decrease as the intramuscular temperature rises above 40°C,^[43] although local temperatures rarely exceed 41 to 42°C in humans, even when exercising in a hot environment.^[60] A more moderate rise of muscle temperature (whether induced by a warm-up or the use of impermeable clothing) decreases muscle viscosity and thus the internal work of muscle contraction,^[61,62] tending to offset any decrease in biochemical efficiency. Mitochondrial adjustments to chronic heat stress may also help to conserve the efficiency of mitochondrial phosphorylation as muscle temperatures rise^[52] [accepting in part mechanism (vii)]. On the other hand, Sawka et al.^[26] found difficulty in postulating a plausible mechanism whereby heat acclimation could improve the overall efficiency of a given muscle fibre. The reduction of intramuscular temperature associated with acclimation is likely to increase viscosity and thus reduce contractile-coupling efficiency. Equally, an acclimation- or training-induced increase of sweat secretion is likely to exacerbate dehydration, again decreasing contractile-coupling efficiency.^[63] Nevertheless, motor unit recruitment patterns can be changed by altering proprioceptive afferent activity.^[64] Sawka et al.^[26] thus speculated that chronic heat exposure might increase proprioceptive afferent activity, leading to a greater recruitment of slow-twitch motor units [accepting mechanism (viii)]. However, it remains unclear to what extent a greater recruitment of slow-twitch motor units and increased biochemical efficiency contribute to the observed decrease in the O_2 cost of physical activity after heat acclimation.

2.1.4 Modifications of Maximal Aerobic Power and Relative Work Intensity by Physical Training and by Heat Acclimation

Unless an athlete is in peak condition, endurance training is likely to improve aerobic fitness,

thereby decreasing the relative intensity of a given bout of exercise, with corresponding decrements in physiological and psychological strain. Heat acclimation yields similar benefits. The decrease in metabolic cost of a given submaximal effort is small, but heat acclimation can also augment maximal oxygen intake ($\dot{V}O_{2max}$), diminishing the relative cost of a given activity.

2.1.5 Resulting Physiological Strain

Some studies^(1,23) have found that increases in core temperature (T_{re}) [as represented by rectal temperature (T_{re}) or esophageal temperature (T_{es})] at a given absolute rate of working are an inverse function of $\dot{V}O_{2max}$. For instance, Shvartz et al.⁽²³⁾ suggested that at the end of 3 hours of exercise in a hot environment the T_{re} (in °C) of young men could be predicted from $\dot{V}O_{2max}$ (in $ml \cdot kg^{-1} \cdot min^{-1}$):

$$T_{re} = 40.15 - 0.03 \cdot \dot{V}O_{2max} \quad (r = -0.65) \quad (Eq. 6)$$

Other authors have seen no consistent relationship between T_{re} and the absolute $\dot{V}O_{2max}$.^(24,65,66) and/or have found that T_{re} is more closely associated with relative exercise intensity (expressed as % of $\dot{V}O_{2max}$).⁽⁶⁵⁻⁶⁹⁾ The use of relative intensity typically removes most of the interindividual variability from the rise of T_{re} during exercise.^(10,70) Davies et al.⁽⁶⁹⁾ suggested that the steady-state T_{re} (in °C) was best described by a quadratic rather than a linear function of % $\dot{V}O_{2max}$:

$$T_{re} = 37.25 - 0.00264 \cdot (\% \dot{V}O_{2max}) + 0.00037 \cdot (\% \dot{V}O_{2max})^2 \quad (Eq. 7)$$

Any factor increasing $\dot{V}O_{2max}$ might thus reduce the T_{re} response at a given metabolic rate. However, if the O_2 cost of a given intensity of exercise and values for the other components of heat exchange remain unchanged, then heat accumulation and T_{re} response should remain unchanged, even if $\dot{V}O_{2max}$ is increased. Sawka et al.⁽⁷¹⁾ found that steady-state core temperatures at a given rate of exercise were not significantly reduced after an unacclimated individual's $\dot{V}O_{2max}$ had been increased passively by autologous erythrocyte infusion. The key change induced by an active, train-

ing-induced increase of $\dot{V}O_{2max}$ is probably an alteration of heat exchange through increased sweating. Aoyagi et al.⁽¹³⁾ noted that endurance training did not change the T_{re} response to a given bout of submaximal exercise if clothing with a limited vapour permeability prevented evaporation of additional sweat. In this regard, Avellini et al.⁽⁷²⁾ reported that if increases of T_{re} were avoided by immersion to the neck in 20°C water during training, then at the end of training T_{re} and mean skin temperature (T_{sk}) were increased for any given submaximal exercise, although HR was decreased. Thus, gains of $\dot{V}O_{2max}$ induced by physical training do not improve heat-exercise tolerance unless there are significant elevations in T_{re} during training sessions⁽⁷²⁻⁷⁴⁾, with resulting increases in the sweat rate induced by a given intensity of effort.^(13,72)

Pandolf et al.⁽⁷⁵⁾ compared responses of young and middle-aged men during 10 days of heat acclimation. Individuals were matched for $\dot{V}O_{2max}$ (52.9 vs 51.3 $ml \cdot kg^{-1} \cdot min^{-1}$), but differed in age by 25 years (21.2 vs 46.4 years) and in running distance by 31 km/wk (7.7 vs 38.8 km/wk). During the first few days of heat acclimation, the middle-aged men had lower final values of T_{re} , T_{sk} and HR, higher rates of sweat secretion, and longer heat-exercise tolerance times than their younger counterparts, but by the tenth day of acclimation there were no intergroup differences of physiological responses or performance times. $\dot{V}O_{2max}$ alone could not be the factor which offered the middle-aged men a physiological advantage; rather, it was their level of regular weekly physical training. Gisolfi et al.⁽⁷⁶⁾ drew similar conclusions from comparisons between distance runners and trained college students (respective mean $\dot{V}O_{2max}$ values 62 vs 60 $ml \cdot kg^{-1} \cdot min^{-1}$). During a 100 minute standard heat-exercise tolerance test, the distance runners maintained significantly lower T_{re} and HR than the college students, although sweat rates were very similar for the 2 groups.

2.1.6 Resulting Psychological Strain

The relative work intensity is an important determinant of perceptual ratings of both physical

exertion^[77-79] and thermal stress.^[80] However, numerous other physiological and neuromuscular influences contribute to the perception of effort and heat,^[81-83] and habituation substantially decreases the discomfort associated with exercise in a hot climate over the first few days of exposure.

The rating of perceived exertion at a given absolute work rate usually decreases with training,^[84-86] although there may be little change in perceptions at a given percentage of $\dot{V}O_{2max}$.^[79] Limitations to this generalisation include the elderly,^[87] patients with exercise-induced symptoms such as angina pectoris,^[77] and individuals wearing protective clothing where sweat can accumulate.^[88]

Training-induced decrements in the relative intensity of exercise are similar whether wearing normal or protective clothing, but ratings of perceived exertion change little after training in the latter condition. Possibly, training does not decrease HR or T_{re} if the exerciser must wear protective clothing, so that cardiac or thermal contributions to the Gestalt of effort perception remain unchanged. Heavily clothed individuals showed increases in T_{sk} (with no change in T_{re}) and increased sweat production (but no change in sweat evaporation) after training.^[12]

Thus, any training-induced reductions in the muscular component of perceived exertion may be offset by negative assessments associated with a hotter, wetter skin surface^[31,32] and a smaller core-to-skin thermal gradient.^[4]

Although heat acclimation does not change the relative intensity of exercise very much, it does decrease ratings of perceived exertion for a lightly clothed person. This trend may reflect a decrease in physiological (HR, T_{re} and T_{sk}) and/or thermal strain, given the close simple (Pearson) and rank (Spearman) correlations between ratings of perceived exertion and ratings of thermal discomfort. In contrast, a period of heat acclimation has no beneficial effect on subjective ratings when a person wears protective clothing while exercising at a moderate intensity.^[12] There is little decrease in HR and any benefit from reductions in T_{re} and T_{sk}

is offset by discomfort associated with a greater accumulation of sweat within the garment.

One study^[12] noted a significant decrease in ratings of perceived exertion and/or thermal discomfort when protected individuals exercised at a low intensity (25% $\dot{V}O_{2max}$); in this experiment, there were reductions in both HR and T_{re} (and an increase in sweat accumulation).

Possibly, skin wettedness and associated cardiovascular strain have more influence than other physiological variables upon the extent of psychological strain, particularly when the individual is wearing protective clothing. A reduction in cardiovascular strain may also be necessary to maximise the benefits from reductions in thermoregulatory strain. Finally, there may be a threshold, beyond which sweat accumulation beneath clothing has a substantial effect on thermal comfort.

2.2 External Power (W)

2.2.1 Methods of Estimating W

During pedalling of a cycle ergometer, external power (W) can be calculated from the power output in watts, preferably with adjustment for frictional losses. During treadmill running or walking, W can be calculated from the treadmill speed (v_s in m/sec) and slope (θ in degrees):^[15,17]

$$W = 3.6 \cdot m \cdot g \cdot (v_s \cdot \sin \theta) \cdot A_D^{-1} \quad (\text{Eq. 8})$$

where m is the total body mass when dressed and/or carrying any load (in kg) and g is the acceleration due to gravity ($9.81 \text{ m} \cdot \text{s}^{-2}$). The term $v_s \cdot \sin \theta$ is the equivalent vertical distance climbed by the individual (in m/sec). W can be calculated in a similar fashion for stepping exercise, but it is much more difficult to estimate for most athletic and occupational tasks.

2.2.2 Modifications of W by Physical Training and by Heat Acclimation

If a specific ceiling value of W is set, either by an athlete's perception of exertion or by laboratory rules regarding HR and T_{re} , the peak performance of external work will show a 10 to 20% increase after training or heat acclimation.

2.3 Convective (C_{res}) and Evaporative (E_{res}) Heat Exchanges through the Respiratory Tract

2.3.1 Methods of Estimating C_{res} and E_{res}

Convective (C_{res}) and evaporative (E_{res}) heat exchanges through the respiratory tract can be calculated from M :^[88]

$$C_{res} = 0.0014 \cdot M \cdot (34 - T_a) \quad (\text{Eq. 9})$$

and

$$E_{res} = 0.0023 \cdot M \cdot (44 - P_a) \quad (\text{Eq. 10})$$

where T_a is the ambient temperature (in °C) and P_a is the ambient water vapour pressure (in torr).

The typical exerciser switches from nasal to oronasal breathing at a ventilation of approximately 35 L/min,^[89] but nevertheless expired gas still attains full saturation at temperatures prevailing in the airway. The chosen values of 34 in Eq. 9 and 44 in Eq. 10 may need changing, depending on the environmental conditions and whether protective clothing is worn. For example, values of 38 in Eq. 9 and 49 in Eq. 10 are more appropriate if the exerciser is in a hot, wet environment and/or is wearing some type of a face mask;^[90] the modified values allow for an average temperature of around 38°C and a corresponding saturated water vapour pressure of around 49 torr in the upper respiratory tract. Given a temperature (T) in the range of 0 to 40°C, the saturated water vapour pressure (P^* in torr) at a given site can be estimated to within 0.02 torr according to the equation of Antoine:^[91]

$$P^* = \exp[18.6686 - 4030.183 \cdot (235 + T)^{-1}] \quad (\text{Eq. 11})$$

Equations 9 and 10 assume a linear relationship between M and ventilation. However, ventilation departs progressively from a linear function of M at intensities above the ventilatory threshold. In situations where the exercise intensity is >60% $\dot{V}O_{2max}$, C_{res} and E_{res} may be estimated using $\dot{V}_E$ (in L/min STPD):^[92]

$$C_{res} = \rho_a \cdot c_{p,a} \cdot (60 \cdot \dot{V}_E) \cdot (\bar{T}_e - T_a) \cdot A_D^{-1} \quad (\text{Eq. 12})$$

and

$$E_{res} = \rho_a \cdot \lambda \cdot (60 \cdot \dot{V}_E) \cdot (W_e - W_a) \cdot A_D^{-1} \quad (\text{Eq. 13})$$

where ρ_a is the density of air (1.169 g/L STPD); $c_{p,a}$ is the specific heat of air (0.001005 kJ · g⁻¹ · °C⁻¹); $\bar{T}_e$ is the time-averaged temperature of expired air (in °C); and λ is the latent heat of vaporisation (2.43 kJ/g). W_e and W_a are the humidity ratios (mass of water per mass of dry air) in expired and ambient air, respectively, calculated as:

$$W_e = 0.622 \cdot [P_e^* \cdot (760 - P_e^*)^{-1}] \quad (\text{Eq. 14})$$

and

$$W_a = 0.622 \cdot [P_a^* \cdot (760 - P_a^*)^{-1}] \quad (\text{Eq. 15})$$

P_e^* and P_a^* being the saturated water vapour pressures (in torr) at the temperatures of expired and ambient air, respectively.

2.3.2 Modifications of C_{res} and E_{res} by Physical Training and by Heat Acclimation

Because the coefficients (0.0014 in Eq. 9 and 0.0023 in Eq. 10) are small, any training- or acclimation-induced reduction in the metabolic cost of exercise has only a minimal effect on the contribution of C_{res} and E_{res} to body heat storage.

2.4 Radiant (R), Convective (C) and Conductive (K) Heat Exchanges at the Skin Surface

2.4.1 Methods of Estimating R, C and K

The combined dry component of heat exchange ($R + C$) can be predicted as:^[6,93]

$$R + C = (F_{cl} \cdot f_{cl} \cdot h) \cdot (\bar{T}_{sk} - T_a) \quad (\text{Eq. 16})$$

F_{cl} is the nondimensional Burton thermal efficiency factor, calculated as:^[94]

$$F_{cl} = (1 + 0.0431 \cdot I_{clo} \cdot f_{cl} \cdot h)^{-1} \quad (\text{Eq. 17})$$

where I_{clo} is the average intrinsic thermal resistance of clothing (expressed in clo units; 1 clo = 0.0431 m² · h · °C · kJ⁻¹), not counting effects from trapped air layers and f_{cl} is the ratio of the surface area of the clothing layer to the Du Bois estimate of skin surface area. The latter is approximated as:

$$f_{cl} = 1.0 + k \cdot I_{clo} \quad (\text{Eq. 18})$$

The factor k varies from 0.15 to 0.35.^[2,6,88,93] h is a combined dry heat transfer coefficient (in $\text{kJ} \cdot \text{m}^{-2} \cdot \text{h}^{-1} \cdot ^\circ\text{C}^{-1}$). It is calculated for the seminude case typical of many athletes, using a combination of radiation (h_r in $\text{kJ} \cdot \text{m}^{-2} \cdot \text{h}^{-1} \cdot ^\circ\text{C}^{-1}$)^[91] and convection (h_c in $\text{kJ} \cdot \text{m}^{-2} \cdot \text{h}^{-1} \cdot ^\circ\text{C}^{-1}$).^[91,95] The average value of h_r is approximated by the relationship:^[91]

$$h_r = 4 \cdot \sigma \cdot \varepsilon \cdot [(\bar{T}_{sk} + \bar{T}_r) \cdot 2^{-1} + 273.15]^3 \cdot (A_r \cdot A_D^{-1}) \quad (\text{Eq. 19})$$

in which σ is the Stefan-Boltzmann constant ($2.04138 \cdot 10^{-7} \text{ kJ} \cdot \text{m}^{-2} \cdot \text{h}^{-1} \cdot \text{K}^{-4}$); ε is the emissivity of the body (0.99); $\bar{T}_{sk}$ is the mean skin temperature (in $^\circ\text{C}$), averaged over an entire session; $\bar{T}_r$ is the mean radiant temperature (in $^\circ\text{C}$), averaged over an entire session; and $A_r \cdot A_D^{-1}$ is the ratio of the effective radiating area to the Du Bois estimate of total body surface area (0.72 to 0.77 when standing, 0.65 to 0.72 when sitting, and 0.67 when crouched).^[5,6,88] The average value of h_c is approximated as:^[91,95]

$$h_c = a \cdot v_s^b + c \cdot v_a^d \quad (\text{Eq. 20})$$

in which v_s and v_a are the speeds of the exerciser and ambient air or water movement, respectively; a or c and b or d are constants and exponents, respectively, that depend on the person's posture and behaviour (for example, $a = 0.00$, $b = 0.00$, $c = 41.86$ and $d = 0.50$ for sitting with air movement; $a = 23.44$, $b = 0.39$, $c = 0.00$ and $d = 0.00$ for indoor treadmill running or walking; $a = 30.98$, $b = 0.53$, $c = 0.00$ and $d = 0.00$ for indoor free running or walking; and $a = 30.98$, $b = 0.53$, $c = 7.12$ and $d = 0.86$ for free running or walking against air movement). Notice that many classical laboratory studies of human responses to heat and exercise have been performed without either the radiant heat or the air movement encountered by the athlete who exercises outdoors, and the importance of these 2 variables during competition has thus tended to be underestimated. T_o is the operative temperature, calculated as:

$$T_o = (h_r \cdot \bar{T}_r + h_c \cdot T_a) \cdot h^{-1} \quad (\text{Eq. 21})$$

Under the conditions of a laboratory climatic chamber, $\bar{T}_r \approx T_a$ and thus $T_o \approx T_a$.

K can be predicted as (adapted from Nishi^[91]):

$$K = (0.0431 \cdot I_{clo})^{-1} \cdot (\bar{T}_{sk} - \bar{T}_{cl}) \cdot (A_c \cdot A_D^{-1}) \quad (\text{Eq. 22})$$

where $\bar{T}_{cl}$ is the mean surface temperature of clothing (in $^\circ\text{C}$), averaged over an entire bout of exercise and $A_c \cdot A_D^{-1}$ is the ratio of the contact area to the Du Bois estimate of the total body surface area. In air, K for the nude individual who is wearing only shoes is negligible. If a part of the clothing surface is in contact with an external object, then the local surface temperature of the clothing becomes equal to the surface temperature of that object.

When a person is immersed in water, both C and K are greatly increased, particularly if the water is stirred by wind, waves or swimming.

2.4.2 Modifications of R , C , and K by Physical Training and by Heat Acclimation

If the temperature of the ambient air exceeds that of the skin surface (for example, when exercising in dry desert heat), an increase of $\bar{T}_{sk}$ decreases dry heat gain (Eq. 16 and Eq. 22). Aoyagi et al.^[13] found that endurance training enhanced the early rise of $\bar{T}_{sk}$ (by a margin of $+1.2^\circ\text{C}$ at rest, decreasing to $+0.5^\circ\text{C}$ after 15 min of moderate exercise at 40°C) when individuals were wearing protective clothing. There was also a parallel trend ($\leq +0.5^\circ\text{C}$) when wearing normal light clothing. Because training did not change T_{re} , it was argued that the enhanced $\bar{T}_{sk}$ reflected a greater early increase of skin blood flow after training.

The potential to develop a large skin blood flow when exercising in the heat depends on an individual's peak cardiac output and their regional distribution of blood flow. The peak cardiac output in turn reflects central blood volume. Although the threshold value of T_c for cutaneous vasodilation is increased by reflexes associated with exercise (through a delay in the activation of the active vasodilator system, rather than through involvement of the active vasoconstrictor system),^[96] this delay is minimised in individuals with expanded blood or plasma volumes.^[97-99] The large blood volume

of the well trained athlete allows a greater skin blood flow for any given T_{e} .

2.4.3 Methods of Estimating Blood and Plasma Volume Change

When estimating exercise- and/or heat-induced changes in blood or plasma volume, the technique of blood sampling must be well controlled.^[9,100] The composition of specimens is greatly altered by: (i) venous stasis following application of a tourniquet; (ii) arm position; (iii) body posture; and (iv) ambient and thus skin temperatures.

Training- or acclimation-related changes in blood volume (ΔBV in %) are indicated simply by the change in haemoglobin concentration (Hb in g/100 ml):^[101]

$$\Delta BV = ([Hb_B \cdot H_A^{-1}] - 1) \cdot 100 \quad (\text{Eq. 23})$$

where the subscripts B and A refer to before and after treatment, respectively. The changes in plasma volume (ΔPV in %) are often estimated from the haematocrit (Hct as a fractional value) and Hb,^[101] or from the Hct alone:^[102]

$$\Delta PV = ([Hb_B \cdot (1 - Hct_A)] \cdot [Hb_A \cdot (1 - Hct_B)]^{-1} - 1) \cdot 100 \quad (\text{Eq. 24})$$

or

$$\Delta PV = [(Hct_B - Hct_A) \cdot Hct_A^{-1}] \cdot (1 - Hct_B)^{-1} \cdot 100 \quad (\text{Eq. 25})$$

The term $(1 - Hct_B)^{-1}$ in Eq. 25 is the proportionality factor that relates red cell volume to plasma volume.^[103]

The estimated magnitude of changes in plasma volume induced by physical training or heat acclimation is similar for the 2 methods of calculation. Notice that it is assumed that there has been no change of red cell volume or mean corpuscular volume over the period of observation.^[104-107] A bout of high-altitude training sometimes induces a temporary increase in the proportion of large, immature erythrocytes.^[108] The estimate based on both Hct and Hb (Eq. 24) may also be preferable to that based on Hct alone (Eq. 25) if prolonged exercise in the heat has led to relatively severe dehydration and a shrinkage of the red cells that

is related to the increase in plasma osmolality.^[105,109,110]

2.4.4 Modifications of Plasma Volume by Physical Training and by Heat Acclimation

Endurance-type physical training and heat acclimation are generally accompanied by a progressive, hormonally regulated expansion of plasma volume.^[111-113] This response is probably mediated by an activation of the arginine-vasopressin and renin-angiotensin-aldosterone systems.^[35,100,114,115] The extent and time course of the plasma volume expansion depends upon: (i) the individual's initial fitness;^[13,25,75] and (ii) the pattern of training or acclimation.^[11,14] Well trained individuals with high initial values of $\dot{V}O_{2\text{max}}$ may show no further increase in plasma volume despite a period of heat acclimation.^[13,25,75] If heat exposure is continued for 2 weeks or more, the early increase of resting plasma volume may also be followed by a secondary return toward pre-acclimation levels.^[11,14] However, the timing of an athlete's acclimation is less critical than might be inferred, since the secondary change does not seem to impair ability to exercise in the heat; possibly, the haemodilution response during exercise is enhanced with acclimation.^[112,113,116,117] The end-result would be that during a bout of vigorous exercise, the same plasma volume would be reached as had been attained at an earlier point in heat acclimation.

The plausibility of enhanced haemodilution depends on the type of exercise that is performed and/or the initial level of hydration of the individual. Transient haemodilution is sometimes caused by an increase of skin temperature upon moving from a cool to a hot environment.^[100] For euhydrated individuals in a hot environment, the usual response from standing rest to treadmill exercise is haemodilution.^[71,113,116,118,119] The possible influence of exercise intensity upon the magnitude of the intravascular fluid shifts is controversial.^[9,100] Exercise-induced haemodilution is probably oncotically mediated, secondary to an increase in the circulating protein content.^[9,11,100] Heat-acclimated individuals who are euhydrated

haemodilute during treadmill exercise in the heat (particularly at intensities below 50% $\text{VO}_{2\text{max}}$).^[71,113,116,118,120] However, if individuals are initially hypohydrated, haemodilution is more limited, and in some cases a loss of circulating protein leads to a decrease of plasma volume from rest to treadmill exercise.^[118,119]

In contrast to treadmill exercise, cycle-ergometer exercise elicits an immediate, hydrostatically mediated haemoconcentration, dependent upon the relationship between mean arterial pressure and relative exercise intensity.^[111,121,122] Changes in mean arterial pressure probably influence capillary hydrostatic pressures,^[122] higher values favouring a net exudation of fluid from the capillary bed to the interstitium.^[9] Heat acclimation accentuates haemoconcentration during cycle-ergometer exercise in the heat, possibly because the plasma volume is initially expanded.^[100] In such situations, a greater efflux of plasma protein may be associated with the greater fluid loss.^[100] In contrast to heat acclimation, physical training has no effect, or even attenuates the reduction in plasma volume when cycling at a given relative intensity of effort.^[111,123] Hypohydration or hypovolaemia also reduces the loss of fluid from the vascular compartment during cycle-ergometer exercise.^[97,124] Conflicting results have been observed when bench-step exercise is performed in a hot environment;^[125] unacclimated individuals seem to haemoconcentrate, whereas heat-acclimated individuals haemodilute.

2.4.5 Mechanisms of Plasma Volume Increase

The suggested mechanisms underlying hypervolaemia^[100] are: (i) an osmotic expansion due to increased intravascular retention of electrolytes; and (ii) an oncotic expansion as a result of increased protein retention.

Endurance training and heat acclimation both increase the ability of the sweat glands to reabsorb sodium (Na^+), possibly by an increased secretion of adrenal corticoids^[126] and/or an altered responsiveness to aldosterone.^[127] Both the Na^+ concentration in sweat at any given rate of sweating and the slope of the relationship between Na^+ concen-

tration and sweat rate are reduced by training or acclimation.^[127,128] However, the view that hypervolaemia can be explained in terms of osmotic expansion may be simplistic,^[100] since investigators^[13,14,112,129,130] have not always found the increase in body mass that would be expected from an osmotic retention of water secondary to enhanced Na^+ conservation by the sweat glands and kidneys.

Haemodilution is associated with a net gain in the total quantity of intravascular protein, so that the protein concentration remains virtually unchanged.^[22,71,112,116-120] The intravascular compartment gains fluid at the expense of the interstitial compartment,^[129] and intercompartmental fluid transfer is responsible for translocation of protein from the interstitium to the circulation.^[100]

The extent of the protein shift may depend on lymph flow.^[118] Hypohydration markedly reduces the interstitial fluid volume,^[131,132] resulting in a smaller volume of more viscous lymph.^[9] If an exerciser develops hypohydration, a reduction of lymphatic flow and the return of less protein to the circulatory system increase interstitial oncotic pressure, facilitating a net outward filtration.^[118]

2.4.6 Effects on Skin Blood Flow

Unless skin blood flow is initially limited by a low ambient or body temperature (a situation only seen if T_{sk} is $< 33.0^\circ\text{C}$),^[13,91] the immediate response of the cutaneous circulation to moderate or vigorous dynamic exercise is a reflex noradrenergic vasoconstriction.^[133] The objectives are presumably to redistribute blood flow to the working muscles and to sustain systemic blood pressure.^[134,135] Thus, both skin blood flow and skin temperature usually fall at the onset of exercise.^[135-137] If physical activity is continued for longer than 5 to 10 minutes, the heat liberated by muscular work induces competing thermoregulatory reflexes, and a net cutaneous vasodilation is initiated.^[96,138] Thereafter, the thermal drive from a rising T_e causes a progressive withdrawal of vasoconstrictor tone.^[139] Skin blood flow is increased, and an increased quantity of body heat is transported to the skin surface. Here, it is eliminated

largely through the evaporation of sweat. However, the increase of skin blood flow is abruptly curtailed when T_{re} reaches a critical value (around 38°C, but varying with such factors as hydration status).^[97,140-142] Little further vasodilation is seen, even if a further increase in T_{re} is permitted.

Opinions concerning the effects of physical training and heat acclimation on skin blood flow during exercise in the heat vary. This is partly because the magnitude of training- or acclimation-related changes in skin blood flow depends on interactions between such conflicting factors as: (i) decreases in the T_{re} threshold for vasodilation; (ii) increases in sweating at any given value of T_{re} with decreases in skin temperature and exacerbated dehydration; (iii) increases in plasma volume; and (iv) decrease in muscle blood flow and total cardiac output at any given work rate (secondary to a widening of arteriovenous O_2 difference).

Vasodilation has been observed at a lower T_{re} after training or acclimation than before.^[11,47] Although the skin blood flow at a given combination of T_{re} and skin temperature is generally higher after training or acclimation, this does not necessarily imply that there will be a greater skin blood flow with any given exercise and heat exposure, since T_{re} and skin temperature are also reduced by training or acclimation.

Some studies have found no increase of sweat rate after training or acclimation (as discussed below). More typically, sweat gland responsiveness is increased by training or acclimation, and in a dry environment this cools the skin more than the core. The core-to-skin thermal gradient is thereby widened, and this allows heat loss to be maintained despite a reduced skin blood flow.^[11,143] (Eq. 26 below). Thirst is not a good guide to dehydration and any increase of sweating may lead to faster dehydration, particularly if the regular intake of readily absorbed fluid is not actively encouraged and/or the rules of competition restrict the drinking of water. Increased sweat loss in turn undoubtedly reduces cutaneous vasodilation and possibly also reduces skin temperature as T_{re} rises.^[14,97,99,144]

Given the latency of sweat secretion and the time needed for sweat to permeate any clothing, training- or acclimation-induced changes in the pattern of sweating do little to alter the skin temperatures of clothed individuals during the first few minutes of exercise in the heat. In such situations, the early fall of skin temperature (which probably reflects a decrease in skin blood flow) seems unaffected by natural acclimatisation to heat.^[137] On the other hand, Aoyagi et al.^[13] saw evidence (an increased T_{sk} with an unchanged T_{re}) suggesting a rise in skin blood flow during moderate-intensity treadmill exercise when the resting plasma volume had increased by some 10% in response to endurance training or heat acclimation. Ohara^[145] estimated that a 1.0°C increase in skin temperature corresponded to an increase in skin blood flow of approximately 25% at an ambient temperature of 30°C and of 40% at 40°C. Assuming that the temperature within the clothing microenvironment was indeed within this range,^[32] the training-induced changes in T_{sk} observed by Aoyagi et al.^[13] (+1.2°C at rest to +0.5°C after 15 minutes of moderate exercise in the heat) imply an early 20 to 30% increase of skin blood flow. Any training-related change in skin flow was progressively attenuated as T_{re} approached 38°C.^[13] The probable reason is that in the later stages of prolonged exercise, the individual had already reached the limit of active vasodilation.^[139]

The transient, early increase of skin blood flow benefits the individual who is wearing clothing with a limited vapour permeability if he or she moves from a relatively cool environment to an environment where the ambient temperature exceeds that of the skin surface. The greater cutaneous blood flow augments convective heat transfer from the deep body tissues for a given core-to-skin thermal gradient (Eq. 26 below), and because of the resulting rise of skin temperature, it limits heat gain by radiation and convection (Eq. 16) and/or conduction (Eq. 22). Core-to-skin heat flow (H_F in $\text{kJ} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$) is expressed as (adapted from Sawka and Wenger^[10]):

$$H_F = [c_{p,M} \cdot (60 \cdot \text{SkBF}) + K_0] \cdot (T_c - \bar{T}_{sk}) \cdot A_D^{-1} \quad (\text{Eq. 26})$$

where $c_{p,b}$ is the average specific heat of the blood (3.85 to $4.19 \text{ kJ} \cdot \text{L}^{-1} \cdot ^\circ\text{C}^{-1}$);^[6,10,146] SkBF is the skin blood flow (in L/min); and K_0 is the thermal conductance of the tissues when skin blood flow is at a minimum (typically 36 to $65 \text{ kJ} \cdot \text{h}^{-1} \cdot ^\circ\text{C}^{-1}$, but varying with body surface area and the thickness of subcutaneous fat).

Aoyagi et al.^[13] suggested that heat acclimation led to some increase in skin blood flow. Although they saw no significant change in $\bar{T}_{sk}$ (and thus no limitation of dry heat gains), there was a significant reduction in T_{re} of 0.2 to 0.3°C after acclimation, irrespective of the type of clothing that was worn. The consequent reduction in core-to-skin thermal gradient implies that there must have been an increase of skin blood flow in order to sustain a given rate of core-to-skin heat transfer and thus to allow an equivalent $\bar{T}_{sk}$ (Eq. 26).

Although skin blood flow during heat-exercise stress increases after both endurance training and heat acclimation, the magnitude of change is greater for training.^[13] Aoyagi et al.^[13] found that the 2 treatments gave a similar 8% expansion of plasma volume, with a negligible increase in evaporative cooling. Compared with heat acclimation, the greater increase of skin blood flow after endurance training may be explained at least in part by less redistribution of blood flow to the working muscles, since training:^[39,70,147]

- maintains a higher haemoglobin concentration and therefore a greater O_2 -carrying capacity of the blood
- results in local increases of enzyme activity which allow a greater extraction of O_2 in the working muscles
- causes an increased capillary supply which offers a more effective distribution of cardiac output to the working muscles.

Blood flow to the viscera and inactive muscles may also decrease.^[7,148]

2.4.7 Effects on Heart Rate (HR)

Perhaps the most consistent and pronounced change associated with either physical training or heat acclimation is a decreased heart rate (HR) during submaximal exercise in the heat.^[11,39,70,149]

Mechanisms include: (i) a decrease in sympathetic drive, as evidenced by a decrease in circulating catecholamines and/or a decreased myocardial sensitivity to catecholamines; (ii) an increase in vagal tone, as evidenced by an increase in plasma acetylcholine concentrations and/or an increased myocardial sensitivity to acetylcholine; (iii) a combination of (i) and (ii); (iv) a decrease in the intrinsic rate of firing of the sinoatrial pacemaker; (v) a more direct effect of the increased blood volume and thus ventricular filling upon stroke volume; (vi) increased venous tone; (vii) decreased peripheral pooling secondary to reduced skin and/or muscle blood flow; and (viii) decreased T_c and skin temperature [secondary to (vi), (vii) and/or increased sweating].

The decrease of sympathetic drive could have a central or a peripheral origin.^[39,150] Endurance training increases myocardial contractility, and (if training is prolonged) there may also be myocardial hypertrophy.^[151,152] The stroke volume for a given ventricular filling is thus increased both at rest and during exercise. The cardiac output of trained individuals is either the same as or slightly less than that of untrained individuals at any given O_2 consumption,^[39,70,149] and given a larger stroke volume, there is much less need for trained individuals to increase their HR. The autonomic outflow to the heart can also be modified by peripheral impulses arising in the muscles and joints and by descending impulses that irradiate from the motor cortex.^[39] A lesser accumulation of vasodilator metabolites in the trained skeletal muscles or a lesser activation of the motor cortex in a trained individual can thus contribute to the slower HR.^[153,154] Such adaptive mechanisms might well account for the effects of long term, intensive training, in contrast to the more limited effects of short term and less intensive training and in particular of heat acclimation. Stroke volume is not increased consistently after heat acclimation;^[11] the total cardiac output at a given rate of work either remains the same (with increased stroke volume) or is reduced (with little change in stroke volume). Heat acclimation also seems unlikely in itself to lead to changes in muscle

biochemistry, particularly the transformation of fast-twitch into slow-twitch muscle fibres and/or alterations within the fast-twitch subgroups reported after strenuous endurance training.^[147] Therefore, an increase in blood volume and/or a decrease in peripheral blood flow may be important factors contributing to the decrease in HR over the course of heat acclimation and short periods of low-intensity training.

In situations where skin and/or muscle blood flow is reduced during exercise and/or heat exposure, the resultant decrease in peripheral pooling augments central blood volume and allows cardiac output to be sustained at a lower HR. Wenger^[11] argued that peripheral venomotor adjustments might play a transitional role in heat acclimation, but surprisingly he found no evidence that such a response made a persistent contribution to a reduction in circulatory strain.

Changes in T_{re} and skin temperature do not provide a satisfactory explanation of HR changes:^[11,13,14]

- the HR at a given T_{re} is the same or greater after acclimation (or training) than before, so body temperatures would need to decrease if the HR were to be lower after acclimation (or training)
- although acclimation lowers T_{re} and skin temperature, it also reduces the T_{re} thresholds for vaso- and venodilation
- exercise HR usually decreases more rapidly than exercise T_{re} over the course of acclimation (or training).

By exclusion, it thus seems likely that an increase in blood volume is the most important factor in reducing HR. In support of this view, Senay et al.^[112] have shown that the time course of the decrease in HR during heat acclimation is roughly similar to that of the increase in plasma volume.

If a person wears protective clothing and/or exercises for a long period at a relatively low intensity, the contribution of reduced peripheral pooling to decreased HR may be increased.^[144] A low intensity of exercise extends the tolerated duration of activity. A larger total volume of sweat can then be evaporated, augmenting overall evaporative cooling, even though the rate of sweat evaporation in

unit time is not increased. In such a situation, the skin temperature is lower for any level of T_{re} , and a given core-to-skin heat transfer is achieved by a smaller skin blood flow (Eq. 26). Cumulative dehydration tends to be more severe with low-intensity, prolonged exercise. This constraint progressively limits the potential for cutaneous vasodilation as T_{re} rises.^[97,99,144] On the other hand, the blood flow demands of the muscles are smaller with a low intensity of exercise. Because skin and/or muscle blood flow are reduced, light activity causes less peripheral pooling, and ventricular filling is better maintained. In this regard, Fortney et al.^[135] indicated that venous pooling might be more important than plasma water loss as a cause of reduced cardiac filling pressures in the heat.

2.4.8 Interaction between Plasma Volume, Skin Blood Flow and HR

Aoyagi et al.^[113] observed that both endurance training and heat acclimation allowed an increase in skin blood flow during the early stages of exercise in the heat. There was no associated change in HR response to the heat-exercise challenge.^[13] These findings suggest that any increase of plasma volume is used to augment skin blood flow, rather than to reduce HR, under conditions where the overall cardiovascular strain is relatively small.

2.4.9 Effects on Sweating and Body Water Loss

Sweat loss reduces total body water content unless an adequate amount of fluid is consumed. If the total volume of sweat secretion is less than 2L, water may be drawn from either the glycogen molecule (in glycogen-depleting activity) or the extracellular compartment (with light exercise under very hot conditions). But as the degree of fluid depletion increases, a progressively greater fraction of the water is drawn from the intracellular space.^[131,132] The reduction in plasma volume bears a general relation to total body water loss, even under severe conditions (exercise + heat + fluid denial).^[119]

Physical training or heat acclimation may modify the relationship of hypovolaemia to overall hypohydration. Sawka et al.^[120] found a smaller reduction of plasma volume for a given decrease in body mass after 10 days of heat acclimation.

Physiological mechanisms could be postulated for this defence of plasma volume, although it might simply reflect an acclimation-induced expansion of plasma volume. Both heat acclimation and physical training increase sweat secretion, and this tends to offset the beneficial effect of plasma volume expansion.^[13]

2.5 Evaporative Heat Loss from the Skin Surface (E_{sk})

2.5.1 Methods of Estimating E_{sk}

Evaporative heat loss from the skin surface (E_{sk}) can be determined from sweat evaporation (SE in g/h):^[15-17]

$$E_{sk} = \lambda \cdot SE \cdot A_D^{-1} \quad (\text{Eq. 27})$$

where λ is the energy equivalent of evaporated sweat (2.43 kJ/g). The weight losses of seminude or dressed individuals are corrected for losses due to exchange of gases (m_r in g/h) and evaporation of water in the respiratory tract (m_e in g/h). The respective values for m_r ^[156] and m_e ^[157] can be estimated as:

$$m_r = (60 \cdot \dot{V}O_2) \cdot (RQ \cdot pCO_2 - pO_2) \quad (\text{Eq. 28})$$

and

$$m_e = 0.019 \cdot (60 \cdot \dot{V}O_2) \cdot (44 - P_a) \quad (\text{Eq. 29})$$

in which pCO_2 and pO_2 are the densities of carbon dioxide (1.977 g/L STPD) and oxygen (1.429 g/L STPD), respectively.

The value of 2.43 kJ/g is the latent heat of vapourisation of water at 30°C. In theory, training- or acclimation-induced changes in the electrolyte content of the sweat,^[127,128,158] the surface temperature of the skin^[13,14] and the water vapour pressure in the microenvironment^[13,14] could all modify the latent heat of evaporation of sweat.^[159] However, in practice, λ appears to be independent of the prevailing air temperature or humidity.^[160,161] Likewise, the usually observed solute concentrations could change the latent heat by no more than 0.01% relative to pure water at any given temperature and humidity.^[161] Various coefficients of λ slightly above 2.50 kJ/g have been reported: 2.60,^[160]

2.61,^[162] 2.70,^[163] 2.81,^[156] 2.89^[164] and 2.91 kJ/g,^[156] although none of these estimates differ significantly from 2.43 kJ/g.^[161] Nevertheless, a change in the assumed value of λ would have a substantial influence on the types of calculation made in this review. It is thus important to initiate research to reach a consensus on an appropriate figure for the latent heat of evaporation of sweat.

E_{sk} may be estimated by an alternative approach,^[93,94] analogous to the method of predicting $R + C$ (Eq. 16). The calculation is based on the characteristics of the clothing, the wetted area of skin, and the saturated water vapour pressure at the observed skin temperature:

$$E_{sk} = (F_{pcl} \cdot f_{cl} \cdot h_a) \cdot (A_w \cdot A_D^{-1}) \cdot (P_{sk}^* - P_a) \quad (\text{Eq. 30})$$

F_{pcl} is the Nishi permeation efficiency factor for a porous clothing assembly, analogous to the Burton thermal efficiency factor F_{cl} (Eq. 17). It is calculated as:

$$F_{pcl} = (1 + 0.0956 \cdot I_{clo} \cdot f_{cl} \cdot h_c)^{-1} \quad (\text{Eq. 31})$$

The constant 0.0956 is the equivalent of a clo unit (in $m^2 \cdot h \cdot ^\circ C \cdot kJ^{-1}$), modified from 0.0397.^[94] h_a is the evaporative heat transfer coefficient (in $kJ \cdot m^{-2} \cdot h^{-1} \cdot torr^{-1}$), expressed as:

$$h_a = 2.2 \cdot h_c \quad (\text{Eq. 32})$$

The coefficient 2.2 is the modified Lewis relation (in K/torr). $A_w \cdot A_D^{-1}$ is the ratio of the wetted skin area to the Du Bois estimate of total surface area (skin wettedness); and P_{sk}^* is the saturated water vapour pressure (in torr) at the mean wetted skin temperature (in °C), averaged over an entire exercise session (see Eq. 11 above).

It is difficult to estimate the skin wettedness factor $A_w \cdot A_D^{-1}$, given its variability both between and within individuals. This affects the prediction of E_{sk} considerably. Heat acclimation and a change of training status further complicate calculations by changing the pattern of sweating responses to a given heat-exercise stress challenge.^[11,46,47,100,165,166]

2.5.2 Modifications of E_{sk} by Physical Training and by Heat Acclimation

E_{sk} generally increases with the volume of sweat secreted, although this depends on whether the environment allows sweat to evaporate and on the amount of sweat that is lost by dripping onto the ground.^[167] Particularly if an athlete is not wearing a singlet, sweat loss by dripping increases rapidly as exercise in a humid climate is prolonged.^[168,169] Physical training and heat acclimation typically increase the sweating response to a given increase of T_{re} ,^[11,46,47,100,166] unless the training has been performed in cool air^[170] or cool water.^[72] Training and acclimation may also alter the distribution of sweating, especially in a humid environment;^[11,165] sweating becomes more uniform across the skin surface, thereby allowing a greater fraction of the secreted sweat to evaporate.

2.5.3 Mechanisms of Increased Sweating

Sweat production can be enhanced if:

- sweating starts at a lower T_c
- the slope relating sweat rate to T_c is steeper
- plateauing of the sweat rate/ T_c relationship occurs at a higher sweat rate.

The changes that occur with training or acclimation may be ascribed to: (i) altered patterns of sweat gland recruitment; and/or (ii) enhanced functioning of previously active sweat glands. Although there are morphologically normal sweat glands that do not normally secrete, these are not necessarily activated by training or acclimation.^[11] Sweat secretion may increase mainly because of increased activity in already functional glands; gland size may be increased,^[171] and/or sensitivity to some trophic factor and/or sympathetic stimulation may be enhanced.^[11]

Training or acclimation does not always increase the volume of sweat secreted in the face of a given environmental challenge.^[13,14,24,25,75] The benefits of a decrease in the threshold T_c for the onset of sweating may be offset by a reduction in T_c either at rest or during exercise. The ability of the trained or acclimated individual to produce more sweat at a given T_c may also be negated by a slower increase of T_c in a given environment, sec-

ondary to a smaller increase in metabolic heat production and/or enhanced evaporative cooling. Finally, any benefit from a potential delay in the plateauing of sweat rate may not be realised because the maximum duration of exercise is predetermined by the rules of competition or by experimental protocol. Other factors that inhibit sweat gland activity include faster dehydration, especially if the intake of fluid is restricted^[9] and maceration of the skin, particularly if the individual is clothed and/or is exercising in a very humid environment.^[166]

2.5.4 Limits of Evaporative Cooling

When a person is clothed, the efficiency of evaporative cooling at the skin surface tends to decrease as the water content of the clothing increases.^[172] Our empirical^[13-16] and statistical data^[17] suggest that the efficiency of evaporative cooling at the skin surface decreases if the sweat evaporation rate exceeds 600 to 650 g/h. Much of the sweat must then accumulate in the clothing and/or evaporate from the clothing surface, the evaporative heat loss is overestimated from the decrease in body mass, and if the heat balance equation is applied, heat storage is underestimated. Once this point has been reached, further sweat secretion has little or no additional cooling effect.^[13-17]

3. Conventional Methods of Estimating Body Heat Storage

Body heat storage is a very important determinant of tolerance of hot environments. Calorimetric solution of the heat balance equation requires climatic chambers with sophisticated instrumentation, available at only a few specialised centres. Accordingly, sports scientists commonly estimate heat storage by a thermometric approach, using equations of the type:^[173]

$$S = (c_{p,b} \cdot Wt \cdot A_D - 1) \cdot (\Delta \bar{T}_b \cdot \Delta t^{-1}) \quad (\text{Eq. 33})$$

where $c_{p,b}$ is the average specific heat of the body tissues ($3.47 \text{ kJ} \cdot \text{kg}^{-1} \cdot ^\circ\text{C}^{-1}$) and $\Delta \bar{T}_b \cdot \Delta t^{-1}$ is the average rate of increase in mean body temperature

(in °C/h) from the beginning to the end of exercise. $\bar{T}_b$ is calculated from measurements of T_c and $\bar{T}_{sk}$:

$$\bar{T}_b = (1 - a) \cdot T_c + a \cdot \bar{T}_{sk} \quad (\text{Eq. 34})$$

where a is a weighting coefficient that depends on skin blood flow^[6] and other specific factors discussed below. In principle, estimates of a vary from 0.10 to 0.33 for vasodilated and vasoconstricted skin, respectively.

3.1 Specific Heat of the Body ($c_{p,b}$)

3.1.1 Sources of Error for $c_{p,b}$

Vallerand et al.^[174] pointed out that differences between calorimetric and thermometric estimates of heat storage could arise from inappropriate assumptions regarding $c_{p,b}$. There is an inter-individual range of 2.93 to 3.77 kJ · kg⁻¹ · °C⁻¹ about the mean value of 3.47 kJ · kg⁻¹ · °C⁻¹.^[173,175] If the lower or upper extreme value were to be used in thermometric calculations, the respective estimates of heat storage would be underestimated by 16% or overestimated by 9%, relative to an estimate based on the average value. Further research is thus required to obtain more precise data on $c_{p,b}$, and factors that are likely to modify values in the athlete, such as lean tissue mass and body fat content.^[176,177]

3.1.2 Modifications of Body Composition and $c_{p,b}$ by Physical Training and by Heat Acclimation

Both physical training and heat acclimation commonly change an individual's body composition and thus $c_{p,b}$.^[39,149,178,179] Training typically decreases fat weight, and leads to slight increases in lean body mass. The magnitude of these changes varies with the intensity, duration and type of activity that is undertaken (particularly aerobic vs strength training), the total daily energy expenditure, and the overall energy balance (distorted in some classes of athlete).

3.1.3 Correction of $c_{p,b}$ for Interindividual Differences in Body Composition

A value of $c_{p,b}$ (in kJ · kg⁻¹ · °C⁻¹) can be calculated for each individual according to his or her fat-free mass and fat mass:

$$c_{p,b} = (1 - \alpha) \cdot c_{p,c} + \alpha \cdot c_{p,s} \quad (\text{Eq. 35})$$

where α is adiposity (mass fraction); $c_{p,c}$ and $c_{p,s}$ are the specific heat capacities of the core fat-free mass (3.72 kJ · kg⁻¹ · °C⁻¹) and the outer fat shell of the body (1.88 kJ · kg⁻¹ · °C⁻¹) respectively.^[180] $c_{p,b}$ only attains a value of 3.47 kJ · kg⁻¹ · °C⁻¹ if the person has between 13.4 and 13.8% body fat. This emphasises the point that most earlier estimates of $c_{p,b}$ were made on average or sedentary young men. Values could be higher for thin athletes (endurance competitors), and are likely to be substantially lower for older men and most women.

3.1.4 Corrections of $c_{p,b}$ for Biological Variation and Thermal State of the Body

The thermometric estimate of heat storage can be calculated taking account of both biological variations in $c_{p,b}$ and a 2-component model of peripheral body temperature change, using equations^[177] of the type:

$$S = (Wt \cdot A_D^{-1}) \cdot [c_{p,c} \cdot (1 - a \cdot \alpha) \cdot (\Delta T_c \cdot \Delta t^{-1}) + c_{p,s} \cdot (b \cdot \alpha) \cdot (\Delta \bar{T}_{sk} \cdot \Delta t^{-1})] \quad (\text{Eq. 36})$$

where a and b are constants that depend on $c_{p,c}$, $c_{p,s}$, and/or the pattern of thermal gradient across the 'shell' of peripheral tissue;^[16] $\Delta T_c \cdot \Delta t^{-1}$ and $\Delta \bar{T}_{sk} \cdot \Delta t^{-1}$ are the rates of increase in core and mean skin temperatures (in °C/h), respectively. Based on the same $c_{p,c}$ and $c_{p,s}$ values as used above, appropriate figures for a and b are 0.66 and 0.33 or 0.75 and 0.50, depending on whether a parabolic or a linear temperature gradient is assumed within the peripheral tissue compartment.

However, Eq. 36 tends to underestimate body heat storage, since the sum of the weighting coefficients for T_c and $\bar{T}_{sk}$ is always < 1.00, irrespective of assumptions regarding patterns of temperature gradient across the 'shell' or the values adopted for $c_{p,c}$ and $c_{p,s}$.^[16] Furthermore, if T_{re} is adopted as representative of T_c , an equation with invariant weighting factors for T_{re} and $\bar{T}_{sk}$ can be used only in situations such as a prolonged bout of exercise, where the contribution of the sluggish, early variation in T_{re} to the total increase in T_{re} becomes negligible.

3.2 Weighting Factors for Local Skin Temperatures

3.2.1 Methods of Determining Local Weighting Factors

$\bar{T}_{sk}$ is calculated as the weighted sum of individual skin temperatures for various body regions. Appropriate weighting factors can be determined on 1 of 3 bases: (i) the percentage of body surface area described;^[173,181] (ii) the thermal sensitivity of a skin region, adjusted for its regional surface area;^[182,183] or (iii) the first eigenvector from a principal component analysis based upon skin temperature determinations at a multiplicity of sites.^[15]

The simple surface-area method is commonly accepted, but thermal receptors are not evenly distributed over the skin surface, and the warming of a sensitive body region has a strong influence on overall perceptions and thermoregulatory effector response.^[10,183] Nadel et al.^[183] based their weightings on changes in thigh sweating rate in response to thermal radiation of selected skin areas. However, the weighing factors thus derived did not differ substantially from weightings based on body surface area alone.^[182]

Interindividual differences in subcutaneous fat distribution may change the contribution of individual thermocouple or thermistor readings to the true $\bar{T}_{sk}$.^[176] Local thermal sensitivity may also be modified by physical training or heat acclimation, depending in part on the type of clothing that is worn during a training programme. Most empirical studies have been performed on seminude individuals under resting conditions. The weightings obtained are thus likely to be inappropriate for exercise, particularly when clothing is worn. Aoyagi et al.^[15] explored the possibility of estimating $\bar{T}_{sk}$ in exercising and clothed individuals, making a principal component analysis of the usually chosen 12-site skin thermistor measurements.

3.2.2 Modifications of Local Weighting Factors by Clothing, Physical Training, Heat Acclimation, and Heat-Exercise Stress

By definition, the weighting factors for the $\bar{T}_{sk}$ calculation determined by the first 2 methods re-

main constant from one situation to another. However, the principal component analysis suggests that the contribution of individual thermistor readings to the first component (which reflects overall $\bar{T}_{sk}$) is modified by the type of clothing worn, by endurance training, heat acclimation, and heat-exercise exposure.^[15] When a person is wearing clothing with a limited vapour permeability, the loading coefficients of 'highly insulated regions' increase as the heat-exercise stress becomes more severe, and the weighting of skin temperatures also becomes more uniform. Of particular note is the change in weighting coefficient of the 'foot' skin temperature for a booted individual. Aoyagi et al.^[15] found that this increased over a succession of exercise sessions in the heat, from 0.00 to 0.01 for the initial bout to 0.10 to 0.12 for the final exposure.

3.2.3 Effects of the Variability of Local Weighting Factors on Mean Skin Temperature and Body Heat Storage Calculations

Aoyagi et al.^[15] found that $\bar{T}_{sk}$ values estimated from regional surface area and principal component methodologies differed from each other by 0.1 to 0.4°C in all experimental conditions except one, significantly so in 19 out of 24 experimental conditions. Nevertheless, the discrepancies (always < 5%) between heat storage estimates based on regional surface area and principal component methodologies had little practical significance.

The small size of differences between the 2 approaches reflects in part the low weighting of $\bar{T}_{sk}$ relative to that of T_{re} in a person who is exercising for a long period in a warm environment. Inter-methodological differences in the coefficients chosen for individual skin regions may also be offset by opposing differences in weighting coefficients for other regions. Mitchell and Wyndham^[184] argued that if $\bar{T}_{sk}$ was calculated from at least 12 measurement points, the average was for practical purposes immune to errors in individual weighting coefficients. Uniformity of skin temperatures is greater in clothed than in nude individuals, and when protective clothing is worn in the heat, skin temperatures become remarkably

uniform.^[4] In such a situation, it may even be sufficient to measure a single lateral or medial thigh temperature (the latter is particularly favoured, since it is unlikely to be influenced by direct impingement of solar or other radiant heat sources).^[4]

3.3 Relative Weightings of Core Temperature (T_c) and Mean Skin Temperature (T_{sk})

3.3.1 Methods of Determining the Relative Weightings of T_c and T_{sk}

Appropriate relative weightings of T_c and T_{sk} have been determined empirically, relating the observed temperature changes to changes in body heat content as calculated by calorimetry.^[15-17,174,180,183,186] Possible approaches include:

- optimisation calculations using stepwise multiple linear regression analysis^[174,180,183,186]
- arbitrary comparisons of proposed weighting systems^[13,16]
- an individual adjustment of weightings based on the person's metabolic rate.^[17]

Stepwise multiple linear regression analysis yields weightings that apply to only a limited range of conditions.^[174,180,183,186] Thus, a variety of formulae have been proposed to estimate T_b from T_c and T_{sk} (coefficients for T_c at the beginning and end of an exercise bout of 0.90/0.90,^[186] 0.87/0.87,^[170] 0.80/0.80,^[173] 0.75/0.75,^[180] 0.70/0.70,^[187] and 0.66/0.79^[183]). No one formula is valid under all conditions. It has yet to be decided whether it is possible to choose a single pair of weighting factors which will give an acceptable approximation of T_b change and body heat storage over a wide range of conditions (for example, <10% error; cf. Mitchell and Wyndham^[184]), rather than seeking optimal weightings for each new set of circumstances. A second possible approach is to use 3 commonly suggested pairs of weighting factors (T_c and T_{sk}) of 2 : 1 (0.67, 0.33), 4 : 1 (0.80, 0.20), and 9 : 1 (0.90, 0.10). These factors give lower, intermediate, and upper estimates of T_b . They provide a representative range, allowing the investigator to carry out a 'sensitivity analysis' that explores the impact of changes in weightings upon

T_b . Aoyagi et al.^[13,16] compared 6 combinations of relative weightings at the beginning and end of prolonged bouts of exercise: 9 : 1 and 9 : 1, 4 : 1 and 4 : 1, 4 : 1 and 9 : 1, 2 : 1 and 2 : 1, 2 : 1 and 4 : 1, or 2 : 1 and 9 : 1. Three other possible sets of weightings (9 : 1 and 2 : 1, 9 : 1 and 4 : 1, or 4 : 1 and 2 : 1) were excluded from their comparisons because they were known to be realistic for cold rather than for hot conditions.^[92,188-190]

Although minor differences are introduced by interindividual and intersport differences in mechanical efficiency, the rate of increase in T_c for any given environment is generally proportional to the heat produced by metabolism, irrespective of the type of exercise that is being performed.^[191,192] This is particularly true if the individual's clothing and/or the environmental conditions restrict evaporative cooling. A third option is thus to use 2 sets of weighting factors, each incorporating an estimate of the individual's metabolic heat production M and its arbitrary level (x in $\text{kJ} \cdot \text{m}^{-2} \cdot \text{h}^{-1}$) that modifies the calculation. Aoyagi et al.^[17] used this tactic, finding appropriate values of x in the following relative weightings of T_c to T_{sk} : $\{4 - [(M - x) \cdot x^{-1}] \cdot 2\} : 1$ for the beginning of his or her exercise bout and $\{4 + [(M - x) \cdot x^{-1}] \cdot 5\} : 1$ for its conclusion; the optimal value of x lay between 450 and 500. When wearing relatively impermeable clothing, the respective relative weightings for the beginning and the end of a 1-hour exercise bout amounted to 4 : 1 and 4 : 1 when the value of M was low ($\leq 20\% \text{VO}_{2\text{max}}$) and $M - x \cdot x^{-1}$ was zero (that is, $M = x$), but extended to 2 : 1 and 9 : 1 when the value of M was moderate (40 to 45% $\text{VO}_{2\text{max}}$) and $(M - x) \cdot x^{-1}$ was 1 (that is, $M = 2 \cdot x$).^[13,16] This approach still needs evaluation both in women and at the higher intensities of exercise common in most types of sport.

3.3.2 Modifications of the Relative Weightings of T_c and T_{sk} according to Clothing, Environment, Metabolic Rate, and Choice of Thermal Model

Many thermal physiologists have adopted an invariant weighting of 4 : 1 for T_c to T_{sk} when estimating the change in T_b under hot conditions (that is, $\Delta T_b = 0.8 \cdot \Delta T_c + 0.2 \cdot \Delta T_{sk}$), whether the

individuals have exercised in shorts^[175,193] or in protective clothing with a low vapour permeability.^[194,195] Aoyagi et al.^[15,16] suggested that this generalisation was true if environmental conditions allowed heavily protected individuals to exercise for 90 minutes or longer (as in some team sports). When wearing protective clothing for periods that were substantially less than 90 minutes, heat storage values predicted from a fixed 4 : 1 weighting of T_{re} and T_{sk} proved to be underestimates relative to values derived from a solution of the heat balance equation. Moreover, the discrepancy between calorimetry and thermometry increased as the rates of rise in T_{re} and T_{sk} became more rapid and the maximum tolerated period of exercise decreased.^[15,16]

Under conditions where clothing places little restriction on the evaporation of sweat, the amount of heat lost through evaporation should increase with metabolic rate. However, when a person is wearing clothing with a limited vapour permeability, the rate of sweat evaporation is relatively constant at low to moderate metabolic rates ($<50\% \text{VO}_{2\text{max}}$),^[13-16] and is actually reduced at higher metabolic rates ($>50\% \text{VO}_{2\text{max}}$).^[196] In the latter circumstance, the heat-exercise tolerance time is too brief to allow water vapour to permeate the overlying clothing and establish the process of evaporation. The rate of sweat evaporation becomes largely independent of the rate of sweat secretion if an individual exercises in clothing with limited vapour permeability.^[13-16,196] These factors may well explain why a fixed 4 : 1 weighting underestimates body heat storage when the severity of thermal conditions preclude prolonged exercise.

The accuracy of thermometric estimates of heat storage can be improved by modifying the weighting factors of T_{re} and T_{sk} according to the environment, the metabolic rate and the choice of model describing the rise of core and peripheral temperatures.^[15-17] However, estimates of heat storage can be distorted by a delayed increase of T_{re} in the first few minutes of exercise, a rapid increase of T_{re} thereafter, and a total increase of $<1.5^\circ\text{C}$.^[15,16] Aoyagi et al.^[16] suggested that this tendency is

particularly marked if the total duration of exercise is ≤ 30 min. The expected increases in T_{re} and T_{sk} are then $\geq 4.0^\circ\text{C/h}$ and $\geq 10.0^\circ\text{C/h}$, values which commonly arise when running outdoors in bright sunshine. One alternative tactic is to use other, more rapidly responding indicators of T_{re} , such as the oesophagus or the tympanic membrane/auditory meatus.^[10] However, such measurements are not convenient when the individual is wearing equipment such as a sports helmet or a respirator.

4. Tolerance of Exercise and Heat Stress

Human tolerance of a combined exercise and heat stress challenge reflects an interaction between the magnitude of physiological and psychological responses and any externally imposed criteria for halting activity. The criteria may be personal, or imposed by an athletic association, occupational health legislation or a human experimentation committee.

4.1 Constraints Imposed by Impermeable and Protective Clothing

A number of factors adversely affect exercise tolerance when a person is wearing protective clothing in the heat. Perhaps the most important constraint is the limited vapour permeability of the clothing. This retards diffusion of water vapour, creating an extremely humid microclimate that thwarts sweat evaporation. The resultant accumulation of sweat within the garment leads to discomfort, dependent on the wetted area of the skin.^[4] Sweat accumulation and body fluid loss are particularly severe when exercising in a hot, humid environment, because the sweat rate, both at rest and during exercise, is linearly related to the moisture content of the air,^[149] at least until the skin becomes macerated. A relatively large fraction of the water that is lost through heavy sweating comes from the blood.^[119,131] Circulatory capacity is thus adversely affected, particularly if water is not replenished frequently. An extra physical load is also imposed upon an athlete or a worker by the mass of any protective equipment that is worn^[33] and by the hobbling that results from multilayered and/or

Table I. Influence of physical training or heat acclimation on selected physiological, psychological and biophysical variables during submaximal, unskilled exercise in the heat while wearing minimal clothing

Variable	Physical training	Heat acclimation
$\dot{V}_E$	↓ or ↔ (for heavy or light exercise)	↓
$\dot{V}O_2$	↔	↓
$\dot{V}CO_2$	↓ or ↔ (for heavy or light exercise)	↓
RQ	↓ or ↔ (for heavy or light exercise)	↓
% $\dot{V}O_{2max}$	↓	↓
Sweat secretion	↑ or ↔	↑ or ↔
Plasma volume	↑	↑
T_{re}	↓ (during exercise)	↓ (at rest and during exercise)
$\bar{T}_{sk}$	↓	↓
HR	↓	↓
Subjective ratings	↓	↓
M	↔	↓
W	↔	↔
C_{res} and E_{res}	↓ or ↔	↓
R, C and K	↓ or ↑ (at $\bar{T}_{sk} > T_a$ or $\bar{T}_{sk} < T_a$)	↓ or ↑ (at $\bar{T}_{sk} > T_a$ or $\bar{T}_{sk} < T_a$)
E_{sk}	↑ or ↔ (in a dry or wet environment)	↑ or ↔ (in a dry or wet environment)
S	↓	↓
Tolerance time	↑	↑

Abbreviations and symbols: C_{res} = rate of convective heat exchange through the respiratory tract; E_{res} = rate of evaporative heat loss from the respiratory tract; E_{sk} = rate of evaporative heat loss from the skin surface; HR = heart rate; M = rate of metabolic heat production; R, C, and K = rates of radiant, convective, and conductive heat exchange through the skin surface; RQ = respiratory quotient; S = rate of body heat storage; T_a = ambient temperature; T_{re} = core temperature; $\bar{T}_{sk}$ = mean skin temperature; $\dot{V}CO_2$ = rate of carbon dioxide production; $\dot{V}_E$ = rate of expiratory ventilation; $\dot{V}O_2$ = oxygen uptake; % $\dot{V}O_{2max}$ = relative work intensity (expressed as percentage of maximal aerobic power); W = rate of external work; ↔ = unchanged value; ↑ = increased value; ↓ = decreased value.

reinforced clothing.^[34] In some occupational settings, a respirator may also impose an external respiratory resistance and dead space.^[197,198] Protective clothing thus exacerbates dehydration and overheating relative to that experienced by lightly clothed individuals who are exercising at a comparable intensity.^[12,199]

4.2 Modifications of the Response to Heat-Exercise Stress Induced by Physical Training and by Heat Acclimation

The benefits of physical training and of heat acclimation for seminude or lightly clothed individuals (see table I) are: (i) improved aerobic fitness and thus a greater cardiovascular reserve (probably developed mainly after training); (ii) a greater potential for heat storage because the initial resting T_{re} is lower (probably seen mainly after acclimation); (iii) a decreased energy cost of exercise [seen after acclimation and also (in athletes) as the learning

component of training]; (iv) an enhanced sweating response at a given percentage of maximal effort (probably found after both treatments); (v) a slower increase in T_{re} due to (iii) and/or (iv) (seen after both treatments); (vi) a reduced HR because of changes in the autonomic nervous system (probably induced mainly by training), expansion of plasma volume (found after both treatments) and/or a decreased peripheral pooling of blood (probably seen after both treatments); and (vii) improved individual tolerance reflecting a decrease in the relative exercise intensity (probably seen mainly after training), a reduction in the physiological strain (found after both treatments) and/or habituation to heat-exercise stress (probably developed by both treatments).

Heavily protected athletes benefit from increased aerobic fitness and lower resting T_{re} (see table II), but gains at moderate intensities of effort are surprisingly small.^[12,13] At low intensities of effort, there may also be a decreased HR and re-

duced perceptual strain.^[12,14] Any acclimation-induced reduction in the metabolic cost of submaximal exercise is small for the person who is wearing protective clothing,^[13,14] because the microenvironment within the protective garment is hot and wet.^[31,32] In this situation, circulatory gains from a training- or acclimation-induced expansion of plasma volume are partially offset by an increase in sweat rate, yet there is little compensation from an increase of evaporative cooling.^[13,14]

4.3 Criteria of Heat-Exercise Tolerance

How can an athlete's tolerance of activity in a hot environment be rated? Many thermal physiologists (for example, Aoyagi et al.^[13,14]) have adopted the following criteria:

- T_c (a ceiling imposed by officials or investigators, for example, 39.3°C in our experiments)

- HR (for example, a ceiling of $\geq 95\%$ of the individual's observed or age-predicted maximum, maintained for 3 min)
- unwillingness of the individual to continue competition or test exercise due to dizziness, nausea, impending loss of consciousness or other reasons (for example, thermal discomfort resulting from a $T_c - \bar{T}_{sk}$ gradient of $< 1.0^\circ\text{C}$ and/or a wetted skin area of $> 40\%$)^[4,200]
- completion of the event or the test bout under conditions predetermined by the rules of competition or experimental protocol.

4.4 Modifications of Heat-Exercise

Tolerance by Physical Training and by Heat Acclimation

The training- or acclimation-induced reduction of physiological and psychological responses to the combined heat-exercise challenge extend the limits imposed by T_c , HR or the individual's

Table II. Influence of physical training or heat acclimation on selected physiological, psychological and biophysical variables during submaximal, unskilled exercise in the heat while wearing protective clothing

Variable	Physical training	Heat acclimation
$\dot{V}_E$	↓ or ↔ (for heavy or light exercise)	↓
$\dot{V}O_2$	↔	↔ or slightly ↓
$\dot{V}CO_2$	↓ or ↔ (for heavy or light exercise)	↓
RQ	↓ or ↔ (for heavy or light exercise)	↓
% $\dot{V}O_{2max}$	↓	↔ or slightly ↓
Sweat secretion	↑	↑
Plasma volume	↑	↑
T_c	↔ or possibly ↓ (for heavy or light exercise)	↓ (at rest and during exercise)
$\bar{T}_{sk}$	↑ (at the early stages of exercise) or ↔	↔ or ↓
HR	↔ or ↓ (for heavy or light exercise)	↔ or ↓ (for heavy or light exercise)
Subjective ratings	↔ or ↓ (for heavy or light exercise)	↔ or ↓ (for heavy or light exercise)
M	↔	↔ or slightly ↓
W	↔	↔
C_{res} and E_{res}	↓ or ↔	↓
R, C and K	↑ or ↓ (at $\bar{T}_{sk} > T_a$ or $\bar{T}_{sk} < T_a$) or ↔	↔ or ↓ or ↑ (at $\bar{T}_{sk} > T_a$ or $\bar{T}_{sk} < T_a$)
E_{sk}	↔ or possibly ↑ (for heavy or light exercise)	↔ or possibly ↑ (for heavy or light exercise)
S	↔ or possibly ↓ (for heavy or light exercise)	↔ or ↓ (for heavy or light exercise)
Tolerance time	↔ or possibly ↑ (for heavy or light exercise)	↔ or ↑ (for heavy or light exercise)

Abbreviations and symbols: C_{res} = rate of convective heat exchange through the respiratory tract; E_{res} = rate of evaporative heat loss from the respiratory tract; E_{sk} = rate of evaporative heat loss from the skin surface; HR = heart rate; M = rate of metabolic heat production; R, C, and K = rates of radiant, convective, and conductive heat exchange through the skin surface; RQ = respiratory quotient; S = rate of body heat storage; T_a = ambient temperature; T_c = core temperature; $\bar{T}_{sk}$ = mean skin temperature; $\dot{V}CO_2$ = rate of carbon dioxide production; $\dot{V}_E$ = rate of expiratory ventilation; $\dot{V}O_2$ = oxygen uptake; % $\dot{V}O_{2max}$ = relative work intensity (expressed as percentage of maximal aerobic power); W = rate of external work; ↔ = unchanged value; ↑ = increased value; ↓ = decreased value.

volition, and should thus increase a person's tolerance of a given set of conditions. This is certainly true of laboratory trials where the individual is seminude or lightly clothed.^[13,14] However, when a person is wearing protective clothing, whether training or acclimation increases tolerance depends on the intensity and thus the maximum duration of the exercise bout,^[13,14] rather than the length of acclimation.^[14] In such circumstances, differences in environmental temperature and humidity have more influence on exercise tolerance at low than at high metabolic rates.^[196]

The tolerance time of the protected exerciser is increased after heat acclimation if exercise is held to a very low intensity (20 to 25% $\text{VO}_{2\text{max}}$) for a long period (100 to 120 minutes).^[14] A similar percentage (about half) of individuals terminate activity voluntarily before the maximum permitted exposure time has been reached at moderate^[13] and low intensities of exercise,^[14] despite a trend for improved subjective ratings in the latter situation.^[12] This might suggest that when wearing protective clothing, the psychological 'end-point' cannot be predicted from perceptual ratings reported over the course of exercise. Nevertheless, such observations do not preclude that reductions in psychological strain contribute to the observed increase in tolerance time. It should be noted that ratings of perceived exertion and thermal discomfort provide different types of information, especially during low-intensity or intermittent exercise. If the intensity of exercise is low and exercise can continue for several hours, Armstrong et al.^[201] found that HR was the only significant predictor of tolerance time.

When wearing protective clothing, the increase in tolerance time (10 to 15%) is similar after 6 and 12 days of heat acclimation.^[14] This seems an important lesson for coaches and sports physicians: tolerance of physical activity can be enhanced by relatively few days of adjustment to a hot environment.

4.4.1 Prediction of Increases in Tolerance Time with Physical Training and/or Heat Acclimation

Assuming that a sports physician permits an increase in T_c of $x^\circ\text{C}$ during a given bout of exercise in the heat, the increase in tolerance time (ΔTT in min) associated with a reduction in resting T_c ($\Delta T_{c,\text{rest}}$ in $^\circ\text{C}$) and a slower rate of increase in T_c ($\Delta T_{c,\text{slope}}$ in $^\circ\text{C/h}$) following training and/or acclimation may be predicted by an equation of the type:^[14]

$$\Delta\text{TT} = -[\Delta T_{c,\text{rest}} + (TT_{\text{initial}} \cdot 60^{-1}) \cdot \Delta T_{c,\text{slope}}] \cdot \{[x \cdot (TT_{\text{initial}} \cdot 60^{-1})^{-1} + \Delta T_{c,\text{slope}}] \cdot 60^{-1}\}^{-1} \quad (\text{Eq. 37})$$

where TT_{initial} is the initial tolerance time (in min).

Let us suppose that an athlete wishes to perform an event lasting 90 minutes, but is initially unable to continue for more than 75 minutes (that is, $TT_{\text{initial}} = 75$). Given that after acclimation the resting T_c is reduced by 0.1°C (that is, $\Delta T_{c,\text{rest}} = -0.1$) and the rate of increase of T_c is slowed by 0.2°C/h (that is, $\Delta T_{c,\text{slope}} = -0.2$), the time from 37.3°C to the 39.3°C ceiling (that is, $x = 2.0$) is extended by about 15 minutes (that is, $\Delta\text{TT} = 15$).

4.5 Interactions of Physical Training and Heat Acclimation

4.5.1 Experimental Protocol to Evaluate the Interaction

To investigate interactions between physical training and heat acclimation, thermal physiologists^[13,46,47] have carried out training under normal ambient conditions, followed by further daily bouts of exercise at high ambient temperatures. Although training in cool conditions might be expected to induce some acclimation, and later exercise sessions in the heat might cause some additional training effects, the bias of such an experimental design is clearly toward training effects first with subsequent acclimation.

4.5.2 Possibility of Further Benefit from Heat Acclimation after Physical Training

Utilising a heterogeneous population ($\text{VO}_{2\text{max}}$ range 35.5 to $63.8 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), Pandolf et al.^[202] suggested that the number of days of heat acclimation needed for T_{re} to reach a plateau during a given bout of heat exposure (Plateau T_{re} Day) was

a function of the individual's initial $\dot{V}O_{2\max}$ (in $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$):

$$\text{Plateau } T_{re} \text{ Day} = 14.49 - 0.165 \cdot \dot{V}O_{2\max} \quad (r = -0.68) \quad (\text{Eq. 38})$$

The implication is that physically fit individuals acclimate to heat more rapidly than persons with a low $\dot{V}O_{2\max}$. By extrapolation, Eq. 38 also implies that well trained individuals with an extremely high $\dot{V}O_{2\max}$ ($> 80 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) are unable to improve physiological and psychological strain after a period of heat acclimation; values are optimal on day 1.

However, the correlation shown in Eq. 38 does not necessarily imply causation. $\dot{V}O_{2\max}$ accounted for only 46% of the variability in the day that T_{re} plateaued during acclimation. Moreover, $\dot{V}O_{2\max}$ may not be the best independent measure to predict differences in heat-exercise tolerance among individuals.^[75,76] Rather the benefits for thermoregulation and cardiovascular function may depend more on the level of regular weekly training than on the value of $\dot{V}O_{2\max}$ *per se*.^[75,76]

Subjects who undertake a good aerobic training programme gain little or no additional benefit from heat acclimation.^[13] Aoyagi et al.^[13] noted that individuals who had been trained by running at 60 to 80% $\dot{V}O_{2\max}$ for 30 to 45 min/day, 3 to 4 days per week for 8 weeks increased their $\dot{V}O_{2\max}$ by 16%. They showed no subsequent changes in resting plasma volume, sweat rate, HR, or maximum tolerated exposure after 6 days of 60 minutes heat acclimation sessions, whether wearing normal or protective clothing. Such observations do not exclude the possibility that highly trained individuals who are already quite tolerant of exercise in warm conditions can prepare themselves further for competition under very adverse conditions by carrying out a training programme in severe heat.^[11]

5. Factors Affecting Physical Training and Heat Acclimation

Several factors can either negate or maximise the circulatory and thermoregulatory advantages

afforded by physical training or heat acclimation.^[9,149,203-212] These include:

- hydration level
- blood doping
- carbohydrate reserves
- electrolyte replacement during competition
- menstrual cycle variations
- circadian rhythms
- sleep deprivation
- gender
- age.

Such factors must be controlled when evaluating the effects of training and heat acclimation.

5.1 Hydration

5.1.1 Hypohydration or Dehydration

Body water loss may result from: (i) administration of diuretics^[97,99,124,213] and other techniques adopted by an athlete who wishes to enter a specific weight category;^[63] (ii) acute thermal-exercise stress;^[13,14,109] or (iii) chronic salt depletion.^[208] The major difference between the first 2 types of water loss is that diuretics cause an isosmotic hypovolaemia, whereas acute thermal stress increases plasma osmolality; this difference influences intravascular fluid shifts, and in the first instance there is a relatively large hypovolaemia for a given reduction in total body water.^[214]

In comparison to normal euhydration, fluid loss prior to and/or during exercise in the heat can cause: (i) impairments of physiological function, including greater increases in T_{re} and HR during the exercise bout;^[119,120,141,215-218] (ii) impairments of muscle function, including reductions in strength and endurance;^[63] and (iii) impairments of physical work capacity.^[213,219] Whether such changes occur depends on the degree of hypohydration and the body compartment that is affected.

The elevated T_{re} in the hypohydrated state is related mainly to a reduction in heat dissipation through both sweating and blood flow to the skin^[99,124,144,215,220] rather than to an increase in metabolic heat production.^[120,215] The relative contribution of attenuated insensible and sensible

heat exchanges to the increase in heat storage depends on environmental conditions. The reduced sweating response is mediated by plasma hyperosmolality^[119,144,216,221] and/or hypovolaemia^[119,124] the former is probably the more important.^[119,144,221] Hyperosmolality may decrease sweating by a direct effect on hypothalamic thermoregulatory centres^[222,223] and/or by a peripheral effect upon the eccrine sweat glands.^[224] Hypovolaemia alters the activity of atrial baroreceptors and thus the afferent neural input to the hypothalamus.^[124] Likewise, the reduction in skin blood flow appears to include both osmotic^[144] and volume loss factors.^[141,225]

A thermal drive from the rising T_{e} activates the vasodilator system and decreases venous tone. The elevated HR in the dehydrated state is thus attributable mainly to a reduced central blood volume and a resultant reduction of stroke volume.^[141,218] Nevertheless, the increase of HR is not always sufficient to compensate fully for the decrease in stroke volume.^[141,218] There is a competition between the needs of the central and peripheral circulations for a limited total blood volume,^[133] and cardiac output decreases below euhydration levels.^[141,218] Cutaneous blood flow is also reduced for a given T_{e} .^[141]

The benefits of training- or acclimation-induced expansion of plasma volume can be reduced or negated, depending on the level of dehydration.^[13,14] On the other hand, there is no convincing evidence that restricting fluid intake during training or acclimation makes an athlete better able to adjust to subsequent work or competition in the heat.^[149] McArdle et al.^[149] have emphasised that a well hydrated athlete always functions at a higher level than one who exercises in a dehydrated state. Because thirst may not provide a good index of body water requirements, *ad libitum* water intake results in incomplete water replenishment when exercising in the heat.^[226-229] Thus, athletes must be urged to rehydrate themselves, according to appropriate guidelines couched for single events in terms of a specified volume intake^[230] or wet bulb globe temperature (WBGT) environmental index^[200] and for

repeated exposures in terms of serum Na^+ change^[205] or body mass loss.^[149]

5.1.2 Hyperhydration: Fluid Replenishment

Given that hypohydration causes a deterioration of performance during heat-exercise stress, can hyperhydration improve performance beyond the levels achieved when a person is euhydrated? Some studies^[99,215,217,231-233] have suggested that ingestion of extra water immediately prior to and/or during exercise in the heat may provide some protection because it delays or even prevents the development of dehydration, increases sweating and skin blood flow, brings about a smaller rise in T_{e} and HR, reduces the perception of effort, and enhances performance during exercise. Other studies^[234] have reported no alteration in sweating rate, T_{e} , HR, and/or endurance time with excessive fluid ingestion. Noakes^[208] has pointed to occasional incidents of hyponatremia and developments of symptoms of diarrhoea and discomfort. Hence, it is important to note that the principal aim of fluid ingestion during exercise is to maintain an appropriate serum osmolality and serum Na^+ concentration, although from the viewpoint of endurance performance a secondary goal is to prevent a decrease in plasma volume.^[208]

The effectiveness of fluid replacement^[149,207] depends on the timing of ingestion, the temperature of the fluid that is consumed, the volume of fluid in the stomach and, possibly, its osmolality or carbohydrate and electrolyte content.

Fluid ingestion during exercise is more effective in maintaining thermal balance than is the consumption of an equal volume of fluid before exercising.^[235] An early report^[236] suggested that cold fluids emptied from the stomach faster than fluids at body temperature, although the importance of fluid temperature has been questioned more recently.^[237,238] Gastric emptying accelerates with gastric volume up to a ceiling of 600ml.^[149] Modest concentrations of glucose may facilitate intestinal transport of water,^[203,234] but gastric emptying is retarded by high concentrations of simple sugars ($>10\%$).^[239-241]

High rates of fluid intake (>1 L/h) are difficult to achieve during exercise, and are likely to lead to abdominal discomfort, possibly due to the accumulation of unabsorbed fluid in the small bowel or colon. Noakes^[208] has pointed out that unless the maximum rate of fluid absorption from the small bowel is known, it is difficult to prescribe how much fluid should be ingested during exercise. The optimum recommendation appears to be the consumption of 400 to 600 ml 20 to 30 minutes before competition, and 150 to 250 ml every 15 minutes during the event; this volume keeps the stomach partially filled, maximising gastric emptying.

5.2 Blood Doping: Artificial Expansion of Plasma Volume

If hyperhydration were to improve performance during heat-exercise stress, improvements would most likely be mediated by hypervolaemia. In fact, some of the thermoregulatory advantages gained through endurance-type training or heat acclimation have been attributed to plasma volume expansion.^[113,114,112] Studies on artificial plasma volume expansion have used infusion of saline or albumin-saline solution^[97,99,124,242-245] and/or reinfusion of autologous blood or erythrocyte-saline solution.^[71,242-247] Regardless of procedure, the expanded plasma volume could reduce cardiovascular and/or thermoregulatory strain when exercising in the heat. However, the effects of intravenous erythrocyte infusion or acute polycythaemia are likely to be greater than those achieved by intravenous saline infusion, since the infusion of whole blood increases an individual's $\dot{V}O_{2\max}$,^[71,242-245] whereas saline infusion may not.^[242-245] The difference in benefit is similar to that seen between endurance training and heat acclimation. Nevertheless, an excessive infusion of red blood cells (and a resulting increase in cellular concentration) could increase blood viscosity, limiting peak cardiac output and thus aerobic power.^[149]

According to Sawka and Young,^[209] the magnitude of the ergogenic effect is also modified by the individual's fitness level and state of heat acclima-

tion. In the person who is not heat-acclimated, an increase of erythrocyte volume leads to compensatory reductions in plasma volume, so that the blood volume remains unchanged.^[71,246] In contrast, if erythrocyte infusion follows heat acclimation, then there is an increase in total circulating protein mass and plasma volume.^[247] For people with high $\dot{V}O_{2\max}$ values, the infusion of erythrocytes and/or saline also increases blood volume.^[243]

5.3 Carbohydrate Replacement

Not only fluid but also carbohydrate depletion can limit exercise performance. It is thus important to decide whether performance in a competitive event is likely to be limited by environmental temperature and thus dehydration or by cumulative glycogen depletion, and to judge the composition of replacement fluids accordingly.

The addition of glucose to ingested fluids increases both fluid and Na^+ absorption,^[248,249] enhancing endurance performance.^[203,204,234,250,251] Beneficial effects on cardiovascular function and thermoregulation have been seen mainly in individuals who have ingested a dilute carbohydrate/electrolyte solution.^[216,234] Glucose polymers have the advantage of maximising carbohydrate content while minimising osmolality.^[240,252-253]

5.4 Electrolyte Replacement

Water loss is always accompanied by some electrolyte loss unless it occurs in the lungs.^[205] Body electrolyte pools are large relative to losses incurred during a single event, and there is no evidence that electrolyte intake during exercise either reduces physiological strain or improves performance relative to the ingestion of water.^[149,230,256] Other factors that sustain mineral balance during and after an event are:

- the hypotonicity of sweat relative to body fluids (sweat glands reabsorb Na^+ by active transport)^[11,257]
- the offsetting of sweat electrolyte loss by the Na^+ -conservation mechanisms of the kidney^[258-261]

- decreases in extracellular volume proportional to any Na⁺ deficit.^[208,228]

Sweat losses of other electrolytes, such as potassium (K⁺), magnesium (Mg²⁺) and calcium (Ca²⁺), are negligible except under extreme conditions.^[131,262-264] There have been suggestions that unacclimatised individuals who are exercising repeatedly in a hot climate need a dietary supplement of 8 to 10g of salt per day,^[11,149] but those who are acclimatised and in a relatively steady state of salt balance may need no Na⁺ supplement beyond that which is contained in a normal diet.^[11,256]

However, others^[205,265,266] have found no evidence that physiological adjustments during heat acclimation are facilitated by a high salt intake relative to a normal diet. A low Na⁺ intake [sodium chloride (NaCl) 5.7 g/day, Na⁺ 98 mEq/day] placed individuals at greater risk of plasma volume deficit, increased T_{re}, and increased HR during the first 3 to 6 days of exercise in the heat compared to a high Na⁺ intake (NaCl 23.2 g/day, Na⁺ 399 mEq/day), but between-diet differences disappeared after 8 days.^[265,266] In contrast, high NaCl diets during 10-day heat acclimation trials (22.5 or 30.0 g/day) increased losses of K⁺, chlorine (Cl⁻), Ca²⁺, and phosphate (PO₄³⁻) relative to a normal diet (15.0 g/day).^[205] These changes caused cardiovascular impairment, with decreased exercise capacity and impaired heat tolerance.^[205] Salt loading possibly contributes to K⁺ depletion, rhabdomyolysis, and heat injury.^[267]

Nevertheless, the addition of a small amount of electrolytes to replacement fluids may improve their flavour, and may allow more complete rehydration compared to water alone.^[221,228,268] If pure water is absorbed from the gut, it can dilute the plasma concentration of Na⁺, stimulating urine production and blunting the Na⁺-dependent stimulation of thirst. Maintaining plasma Na⁺ concentration by adding a small amount of this electrolyte to the ingested fluid may sustain the thirst drive, so that lost plasma volume is restored more rapidly. This may lead in turn to a better regulation of plasma tonicity. Thus, the increased plasma concentrations of fluid- and electrolyte-regulating

hormones such as vasopressin and the renin-angiotensin-aldosterone axis that develop during prolonged exercise^[246,269] are controlled better by the ingestion of Na⁺-containing solutions than by pure water.^[270-272]

5.5 Menstrual Cycle

In women, the resting T_e is approximately 0.4°C higher during the luteal than during the follicular phase of the menstrual cycle.^[211,212] This reflects the actions of progesterone,^[273,274] oestrogen,^[146,275] and possibly cytokines such as interleukin-1 (IL-1).^[276] The threshold T_e for the onset of thermoregulatory responses such as sweating and vasodilation is increased during the luteal phase.^[273,277,278] On the other hand, the slope of the relationship of the effector response to T_e seems unchanged between menstrual phases. There may also be hormonally mediated differences in fluid volume homeostasis between different phases of the menstrual cycle.^[211,212]

It is thus important when evaluating responses to exercise in the heat that observations be made in the same phase of menstruation. Many reports^[279-282] have suggested that the menstrual cycle has little effect on actual performance in the heat. Unfortunately, the critical defended variable (resting T_e)^[273,277,278] was overlooked in many such studies.^[279,280,282] Recent investigations^[274,283] suggest that women have more difficulty in reaching thermal equilibrium if they exercise in the luteal phase.

5.6 Circadian Rhythms

Circadian rhythms in T_e, skin blood flow, skin temperature, and HR are similar in magnitude and phase, showing higher values in the afternoon and lower values in the morning.^[211] The rhythm of T_e must reflect circadian changes in heat production, heat loss, or both.^[284,285] Heat production in itself does not explain all of the circadian rhythm, as shown by desynchronisation of the activity and T_e cycles.^[286,287] The regulation of T_e, as determined by the threshold changes for sudomotor and vasomotor responses, appears to follow a similar circa-

dian pattern to that for T_{e} .^[288,289] Moreover, this trend is not influenced by the menstrual cycle.^[278]

5.7 Sleep Deprivation

Bursts of sweating occur during slow wave sleep, owing to a temporary reduction in the thermoregulatory 'set point'.^[211] Rapid eye movement (REM) sleep, in contrast, inhibits the sudomotor response to a given thermal drive, without altering thermoregulatory vasomotor responses, so that T_{a} and T_{e} increase.^[290,291]

Resting T_{e} is reduced during sleep deprivation,^[292,293] but indices of performance such as $\text{VO}_{2\text{max}}$ show little change.^[206,210] The lower initial T_{e} increases the potential for total body heat storage and thus extends potential heat tolerance (Eq. 37). However, an extended loss of sleep (> 30h) attenuates effector responses for heat dissipation.^[292,294] The predominant effect of sleep loss is a decrease in the slope of both sweating and cutaneous blood flow/ T_{e} relationships, with no change in the T_{e} threshold for their onset. Possible peripheral alterations may include: (i) a decrease in the concentration of neurotransmitter *per* sudomotor impulse; (ii) a decrease in the cholinergic sensitivity of the sweat gland; and/or (iii) a decrease in blood flow to the gland. An attenuated vasodilatory response indicates a peripheral modulation of effector mechanisms similar to that observed for sweating.^[292] It cannot be explained in terms of blood volume regulation, since an expansion of plasma volume is generally observed,^[206,295] secondary to acidosis or changes in diet.^[210] Moreover, plasma renin activity is depressed as a result of long term postural and blood volume changes, and this should result in less rather than more vasoconstrictor activity.^[210]

Regardless of precise mechanisms, sleep loss increases the rise of T_{e} for a given metabolic rate,^[293] offsetting or negating any gain from a lower resting T_{e} .

5.8 Gender and Age

Other factors such as gender^[263,296-298] and age^[299-301] can influence physiological responses

to exercise and heat exposure. However, the effects of both gender and age are less pronounced if studies are controlled for $\text{VO}_{2\text{max}}$ (which independently affects thermoregulation). It remains unclear whether individuals should be equated in terms of absolute $\text{VO}_{2\text{max}}$, or their status relative to age- and gender-specific population norms. Women (especially in the follicular phase of the menstrual cycle)^[302,303] tolerate the physiological and thermal stress of exercise at least as well as men of comparable acclimatisation and $\text{VO}_{2\text{max}}$.^[279,280-282,302,304-306] The same is true of elderly individuals who have been matched for absolute aerobic power,^[75,307,308] although a diminished thermoregulatory ability remains an important characteristic of the aging process.^[299,301]

Nevertheless, when compared to adult men, adult women^[298,306] and children^[309] rely more heavily on dimensional characteristic and/or circulatory mechanisms for heat dissipation to compensate for a lesser sweat production. This places women and children at a disadvantage in very hot environments, since their smaller body mass-to-surface area ratio increases their rate of body heating. The lower sweat output may make women and children less vulnerable to dehydration in the heat;^[149] however, this potential advantage is probably offset by their smaller volume of body fluids.

6. Practical Recommendations to Coaches and Sports Physicians

Physical training and heat acclimation lead to similar improvements in exercise tolerance under hot conditions. Gains include an increased blood volume, increased sweat production, and decreased thermal discomfort. Nevertheless, there are differences between the 2 forms of preparation. Physical training is associated with an increased aerobic fitness and a decrease in relative work intensity, whereas heat acclimation leads to a lowered thermoregulatory 'set point' and a reduced energy cost for a given task. Maximal adjustment to work in a hot environment requires that competitors be both well trained and fully heat-acclimated.

6.1 Planning of Physical Training Programmes

The training component of preparation for exercise in the heat follows the standard principles of exercise prescription.^[39,149] The response depends on:

- initial fitness level
- intensity of exercise
- duration of exercise
- frequency of exercise
- type of exercise.

Intensity plays a principal role in stimulating the body's cardiovascular and thermoregulatory apparatus, although additional gains are small if training intensities exceed 85% $\dot{V}O_{2max}$.^[13,24,39,47,74,149] Comparisons of distance runners and trained college students suggest that optimal results are obtained by long-duration training sessions at or near the ceiling intensity.^[76]

6.2 Heat Acclimation and Its Planning

The optimal method of acclimatisation to a hot environment is to spend several weeks in that environment. However, if this is not possible, some advantage can be gained from artificial acclimation. The extent of the acclimation-induced physiological and psychological changes depends on:

- initial acclimatisation and fitness level
- the severity of the heat stress presented during acclimation sessions
- inclusion of exercise during acclimation sessions
- the length of individual sessions.

Each exposure must be sufficient to raise T_{re} by a substantial amount and to provoke moderate to profuse sweating; heat exposure is most effective when combined with exercise. Moderate exercise in the heat for an hour a day enhances the status of unacclimatised individuals, but in a fit and partially acclimatised athlete a similar (or somewhat shorter) period of effort at the maximum tolerated intensity is required in order to improve performance further.^[13,25] Changes develop rapidly over

the first few days of acclimation, and are essentially complete after 14 days.^[11,114] Acclimation seems to have 2 successive phases:^[11,114] (i) early cardiovascular changes that increase plasma volume, reduce HR and habituate the autonomic nervous system, redirecting the available cardiac output to the skin and active muscles; and (ii) late thermoregulatory changes that progressively increase sweating and decrease skin blood flow. Thus, an optimal plan for an endurance athlete who will compete in the heat might involve moderately vigorous exercise (up to 75% $\dot{V}O_{2max}$) in the heat (40 to 50°C), 60 to 120 min/day for 1 to 2 weeks. If an environmental chamber is not available, one alternative is to exercise while wearing clothing with a limited vapour permeability.^[310] The hot and wet microenvironment within such clothing stimulates the body's thermoregulatory apparatus, even though the ambient temperature may be relatively low. Improvements in the ability to maintain a high sweat rate under humid conditions can be greater after acclimation to wet heat than acclimation to dry heat.^[11] Under humid conditions, acclimation reduces the tendency to maceration of the skin and makes sweating more uniform across the skin surface;^[11] this maximises use of the skin or clothing surface for evaporation of sweat.

Acclimatisation in a natural environment differs from laboratory acclimation; in the natural environment, periods of heat exposure are more prolonged (usually for much of the daylight hours), and the person learns to adjust to the total environment. Full adjustment to a hot environment may take months or even years. In general, people residing in tropical climates have, as a result of natural acclimatisation, the same sorts of responses to exercise in the heat as are acquired through a programme of artificial acclimation,^[11] but they also develop more slowly acquired behavioural changes. Nevertheless, although better acclimatised than their temperate counterparts in many respects,^[311] naturally acclimatised persons often need exposure to severe heat-exercise stress in order to match the heat tolerance that has been devel-

oped by athletes who have undergone rigorous artificial acclimation.^[11]

7. Conclusions

As little as 1 to 2 weeks of physical training and artificial heat acclimation can improve a lightly clothed person's homeostatic mechanisms, reducing the physiological and psychological strains imposed by a given combination of exercise and heat stress. However, if athletes wear protective clothing with limited vapour permeability, the benefits of either acclimation or an increase of training are relatively small. Training and/or acclimation can do little to improve the heat tolerance of protected persons if an event demands high intensities of exercise. If rules permit, frequent changes of players may be helpful in team sports.

Acknowledgements

Dr Roy J. Shephard is supported as a CTAL Resident Scholar in the Health Studies Programme, Brock University, St Catharines, Ontario, Canada

References

- Armstrong LE, Pandolf KB. Physical training, cardiorespiratory physical fitness and exercise-heat tolerance. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. Human performance physiology and environmental medicine at terrestrial extremes. Indianapolis: Benchmark, 1988: 199-226
- Gagge AP, Nishi Y. Heat exchange between human skin surface and thermal environment. In: Lee DHK, editor. Handbook of physiology: reactions to environmental agents. Bethesda: American Physiological Society, 1983: 69-92
- Gisolfi CV, Wenger CB. Temperature regulation during exercise: old concepts, new ideas. In: Terjung RL, editor. Exercise and sport sciences reviews. New York: Macmillan, 1984; 12: 339-72
- Goldman RF. Heat stress in industrial protective encapsulating garments. In: Levine SP, Martin WF, editors. Protecting personnel at hazardous waste sites. Boston: Butterworth, 1985: 215-66
- Gonzalez RR. Biophysics of heat transfer and clothing considerations. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. Human performance physiology and environmental medicine at terrestrial extremes. Indianapolis: Benchmark, 1988: 45-95
- Parsons KC. Human thermal environments: the effects of hot, moderate and cold environments on human health, comfort and performance - the principles and the practice. London: Taylor & Francis, 1993
- Rowell LB. Human cardiovascular adjustments to exercise and thermal stress. *Physiol Rev* 1974; 54: 75-159
- Santee WR, Gonzalez RR. Characteristics of the thermal environment. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. Human performance physiology and environmental medicine at terrestrial extremes. Indianapolis: Benchmark, 1988: 1-43
- Sawka MN. Body fluid responses and hypohydration during exercise-heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. Human performance physiology and environmental medicine at terrestrial extremes. Indianapolis: Benchmark, 1988: 227-66
- Sawka MN, Wenger CB. Physiological responses to acute exercise-heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. Human performance physiology and environmental medicine at terrestrial extremes. Indianapolis: Benchmark, 1988: 97-151
- Wenger CB. Human heat acclimatization. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. Human performance physiology and environmental medicine at terrestrial extremes. Indianapolis: Benchmark, 1988: 153-97
- Aoyagi Y. Endurance training, heat acclimation, and protective clothing: the thermophysiology of exercising in a hot climate [dissertation]. Toronto: University of Toronto, 1996
- Aoyagi Y, McLellan TM, Shephard RJ. Effects of training and acclimation on heat tolerance in exercising men wearing protective clothing. *Eur J Appl Physiol* 1994; 68: 234-45
- Aoyagi Y, McLellan TM, Shephard RJ. Effects of 6 versus 12 days of heat acclimation on heat tolerance in lightly exercising men wearing protective clothing. *Eur J Appl Physiol* 1995; 71: 187-96
- Aoyagi Y, McLellan TM, Shephard RJ. Determination of body heat storage in clothing: calorimetry versus thermometry. *Eur J Appl Physiol* 1995; 71: 197-206
- Aoyagi Y, McLellan TM, Shephard RJ. Determination of body heat storage: how to select the weighting of rectal and skin temperatures for clothed subjects. *Int Arch Occup Environ Health* 1996; 68: 325-36
- Aoyagi Y, McLellan TM, Shephard RJ. Residual analysis in the determination of factors affecting the estimates of body heat storage in clothed subjects. *Eur J Appl Physiol* 1996; 73: 287-98
- Shephard RJ. Exercise physiology. Toronto: Decker, 1987
- Du Bois D, Du Bois EF. Clinical calorimetry: a formula to estimate the approximate surface area if height and weight be known. *Arch Intern Med* 1916; 17: 863-71
- Morgan WP. Hyperventilation syndrome: a review. *Am Ind Hyg Assoc J* 1983; 44: 685-9
- Shephard RJ. Learning, habituation, and training. *Int Z Angew Physiol* 1969; 28: 38-48
- Senay LC, Kok R. Effects of training and heat acclimatization on blood plasma contents of exercising men. *J Appl Physiol* 1977; 53: 591-9
- Shvartz E, Shapiro Y, Magazanik A, et al. Heat acclimation, physical fitness, and responses to exercise in temperate and hot environments. *J Appl Physiol* 1977; 43: 678-83
- Gisolfi CV. Work-heat tolerance derived from interval training. *J Appl Physiol* 1973; 35: 349-54
- Houmard JA, Costill DL, Davis JA, et al. The influence of exercise intensity on heat acclimation in trained subjects. *Med Sci Sports Exerc* 1990; 22: 615-20
- Sawka MN, Pandolf KB, Avellini BA, et al. Does heat acclimation lower the rate of metabolism elicited by muscular exercise? *Aviat Space Environ Med* 1983; 54: 27-31

27. Young AJ, Sawka MN, Levine L, et al. Skeletal muscle metabolism during exercise is influenced by heat acclimation. *J Appl Physiol* 1983; 59: 1929-35
28. King DS, Costill DL, Fink WJ, et al. Muscle metabolism during exercise in the heat in unacclimatized and acclimatized humans. *J Appl Physiol* 1983; 59: 1350-4
29. Kirwan JP, Costill DL, Kuipers H, et al. Substrate utilization in leg muscle of men after heat acclimation. *J Appl Physiol* 1987; 63: 31-5
30. Wyndham CH, Rogers OG, Sensy LC, et al. Acclimatization in a hot, humid environment: cardiovascular adjustments. *J Appl Physiol* 1976; 40: 779-85
31. Kakitubata N, Osei K, Michon H, et al. Dynamic moisture permeation through clothing. *Aviat Space Environ Med* 1988; 59: 49-53
32. Sullivan PJ, Mekjavic IB. Temperature and humidity within the clothing microenvironment. *Aviat Space Environ Med* 1992; 63: 186-92
33. Duggan A. Energy cost of stepping in protective clothing ensembles. *Ergonomics* 1988; 31: 3-11
34. Teitlebaum A, Golzman RF. Increased energy cost with multiple clothing layers. *J Appl Physiol* 1972; 32: 743-4
35. Francesconi RP. Endocrinological responses to exercise in stressful environments. *Exerc Sport Sci Rev* 1988; 16: 255-84
36. Graesser DK. Thyroid hormones. In: Murray RK, Graesser DK, Mayes PA, et al., editors. *Harper's biochemistry*. 21st ed. Norwalk: Appleton & Lange, 1988: 496-501
37. Winder WW. Time course of the T₃- and T₄-induced increase in rat soleus muscle mitochondria. *Am J Physiol* 1979; 236: C132-8
38. Srikanth K, Sawhney RC, Mathew L, et al. Thyroid gland function during cross adaptation to heat and cold in man. *Int J Biometeor* 1986; 30: 223-30
39. Fox EL, Mathews DK. The physiological basis of physical education and athletics. 3rd ed. Philadelphia: Saunders, 1981
40. Shephard RJ. The oxygen cost of breathing during vigorous exercise. *Q J Exp Physiol* 1966; 51: 336-50
41. Rodwell VW. Enzymes: kinetics. In: Murray RK, Graesser DK, Mayes PA, et al., editors. *Harper's biochemistry*. 21st ed. Norwalk: Appleton & Lange, 1988: 61-74
42. Zachariassen KE. Hypothermia and cellular physiology. *Arctic Med Res* 1991; 50 Suppl. 6: 13-7
43. Brooks GA, Hittelman KJ, Faulkner JA, et al. Temperature, skeletal muscle mitochondrial functions, and oxygen debt. *Am J Physiol* 1971; 220: 1053-9
44. Brooks GA, Hittelman KJ, Faulkner JA, et al. Tissue temperatures and whole-animal oxygen consumption after exercise. *Am J Physiol* 1971; 221: 427-31
45. Shvartz E, Saar E, Meyerstein N, et al. Heat acclimatization while wearing vapour-barrier clothing. *Aerosp Med* 1973; 44: 609-12
46. Nadel ER, Pandolf KB, Roberts MF, et al. Mechanisms of thermal acclimation to exercise and heat. *J Appl Physiol* 1974; 37: 515-20
47. Roberts MF, Wenger CB, Stolwijk JAJ, et al. Skin blood flow and sweating changes following exercise training and heat acclimation. *J Appl Physiol* 1977; 43: 133-7
48. Dimari GP, Malhotra MS, Sen Gupta J, et al. Alterations in aerobic-anaerobic proportions of metabolism during work in heat. *Eur J Appl Physiol* 1980; 45: 43-50
49. Nielsen B, Savard G, Richter EA, et al. Muscle blood flow and muscle metabolism during exercise and heat stress. *J Appl Physiol* 1990; 69: 1040-6
50. Savard GK, Nielsen B, Laszczynska J, et al. Muscle blood flow is not reduced in humans during moderate exercise and heat stress. *J Appl Physiol* 1988; 64: 649-57
51. Smolander J, Kolarik P, Korhonen O, et al. Aerobic and anaerobic responses to incremental exercise in a thermoneutral and a hot dry environment. *Acta Physiol Scand* 1986; 128: 15-21
52. Young AJ. Energy substrate utilization during exercise in extreme environments. *Exerc Sport Sci Rev* 1990; 18: 65-117
53. Bylund A-C, Björk T, Cederblad G, et al. Physical training in men: skeletal muscle metabolism in relation to muscle morphology and running ability. *Eur J Appl Physiol* 1977; 36: 151-69
54. Jansson E, Kaijser L. Substrate utilization and enzymes in skeletal muscle of extremely endurance-trained men. *J Appl Physiol* 1987; 62: 999-1005
55. Karlsson J, Nordstam L-O, Saltin B. Muscle glycogen utilization during exercise after physical training. *Acta Physiol Scand* 1974; 90: 210-7
56. Shephard RJ. *Biochemistry of physical activity*. Springfield: Thomas, 1984
57. Gassner GA, Brooks GA. Muscular efficiency during steady-state exercise: effects of speed and work rate. *J Appl Physiol* 1975; 38: 1132-9
58. Whipp BJ, Wasserman K. Efficiency of muscular work. *J Appl Physiol* 1969; 26: 644-8
59. Sawka MN, Petrofsky JS, Phillips CA. Energy cost of submaximal isometric contractions in cat fast and slow twitch muscles. *Pflügers Arch* 1981; 390: 164-8
60. Saltin B, Gæge AP, Stolwijk JAJ. Muscle temperature during submaximal exercise in man. *J Appl Physiol* 1968; 25: 679-88
61. Blomstrand E, Bando-Ostergaard B. Influence of reduced muscle temperature on metabolism in type I and type II human muscle fibres during intensive exercise. *Acta Physiol Scand* 1987; 131: 569-74
62. Kruk B, Pekkarinen H, Harri M, et al. Thermoregulatory responses to exercise at low ambient temperature performed after precooling or preheating procedures. *Eur J Appl Physiol* 1990; 59: 416-20
63. Fogelholm M. Effects of bodyweight reduction on sports performance. *Sports Med* 1994; 18: 249-67
64. Grimby L, Hannerz J. Disturbances in voluntary recruitment order of low and high frequency motor units on blockades of proprioceptive afferent activity. *Acta Physiol Scand* 1976; 96: 207-16
65. Drinkwater BL, Denice JE, Kappert IC, et al. Aerobic power as a factor in women's response to work in hot environments. *J Appl Physiol* 1976; 41: 815-21
66. Greenleaf JE, Castle BL, Ruff WK. Maximal oxygen uptake, sweating and tolerance to exercise in the heat. *Int J Biometeor* 1972; 16: 375-87
67. Davies CTM. Influence of skin temperature on sweating and aerobic performance during severe work. *J Appl Physiol* 1979; 47: 770-7
68. Davies CTM. Thermoregulation during exercise in relation to sex and age. *Eur J Appl Physiol* 1979; 42: 71-9

69. Davies CTM, Brotherhood JR, Zaidifard E. Temperature regulation during severe exercise with some observations on effects of skin wetting. *J Appl Physiol* 1976; 41: 772-6
70. Åstrand P-O, Rodahl K. Textbook of work physiology: physiological bases of exercise. 3rd ed. New York: McGraw-Hill, 1986
71. Sawka MN, Dennis RC, Gonzalez RR, et al. Influence of polycythemia on blood volume and thermoregulation during exercise-heat stress. *J Appl Physiol* 1987; 62: 912-8
72. Avellani BA, Shapiro Y, Fortney SM, et al. Effects on heat tolerance of physical training in water and on land. *J Appl Physiol* 1982; 53: 1291-8
73. Gisolfi CV, Cohen JS. Relationships among training, heat acclimation, and heat tolerance in men and women: the controversy revisited. *Med Sci Sports* 1979; 11: 56-9
74. Pandolf KB. Effects of physical training and cardiorespiratory physical fitness on exercise-heat tolerance: recent observations. *Med Sci Sports* 1979; 11: 60-5
75. Pandolf KB, Cadorette BS, Sawka MN, et al. Thermoregulatory responses of middle-aged and young men during dry-heat acclimation. *J Appl Physiol* 1988; 65: 65-71
76. Gisolfi CV, Wilson NC, Claxton B. Work-heat tolerance of distance runners. In: Milvy P, editor. The marathon: physiological, medical, epidemiological, and psychological studies. New York: The New York Academy of Sciences, 1977: 139-50
77. Borg GAV. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982; 14: 377-81
78. Kamon E, Pandolf K, Cafarelli E. The relationship between perceptual information and physiological responses to exercise in the heat. *J Hum Ergol* 1974; 3: 45-54
79. Sargent AJ, Davies CTM. Perceived exertion during rhythmic exercise involving different muscle masses. *J Hum Ergol* 1973; 2: 3-11
80. Hollies NRS. Psychological scaling in comfort assessment. In: Hollies NRS, Goldman RF, editors. Clothing comfort: interaction of thermal, ventilation, construction and assessment factors [proceedings of the Fibre Society, Inc. Comfort Symposium]. Ann Arbor: Ann Arbor Science, 1977: 107-20
81. Carton RL, Rhodes EC. A critical review of the literature on ratings scales for perceived exertion. *Sports Med* 1983; 2 (3): 198-222
82. Follenius M, Brandenberger G, Oyono S, et al. Cortisol as a sensitive index of heat-intolerance. *Physiol Behav* 1982; 29: 509-13
83. Shephard RJ, Vandewalle H, Bouchel VGE, et al. Respiratory, muscular, and overall perceptions of effort: the influence of hypoxia and muscle mass. *Med Sci Sports Exerc* 1992; 24: 556-67
84. Kneutgen HG, Nordenjö L-O, Ollander B, et al. Physical conditioning through interval training with young male adults. *Med Sci Sports* 1973; 5: 220-6
85. Lewis S, Thompson P, Areakog N-H, et al. Transfer effects of endurance training to exercise with untrained limbs. *Eur J Appl Physiol* 1980; 44: 25-34
86. Pandolf KB, Burne RL, Goldman RF. Differentiated ratings of perceived exertion during physical conditioning of older individuals using leg-weight loading. *Percept Mot Skills* 1975; 40: 563-74
87. Sidney KH, Shephard RJ. Perception of exertion in the elderly, effects of aging, mode of exercise and physical training. *Percept Mot Skills* 1977; 44: 999-1010
88. Fanger PO. Thermal comfort: analysis and applications in environmental engineering. Copenhagen: Danish Technical Press, 1970
89. Niinimä V, Cole P, Mintz S, et al. The switching point from nasal to oronasal breathing. *Respir Physiol* 1980; 42: 61-71
90. Livingstone SD, Nolan RW, Cain JB, et al. Effect of working in hot environments on respiratory air temperatures. *Eur J Appl Physiol* 1994; 69: 98-101
91. Nishi Y. Heat exchange between human body and environment [in Japanese]. In: Nakayama T, editor. Thermophysiology. Tokyo: Rikōgakusha, 1981: 33-72
92. Tikalsis P, McCracken DH, Radomski MW. Heat debt during cold air exposure before and after cold water immersions. *J Appl Physiol* 1991; 71: 60-8
93. Oohori T, Berglund LG, Gagge AP. Comparison of current two-parameter indices of vapour permeation of clothing - as factors governing thermal equilibrium and human comfort. *ASHRAE Trans* 1984; 90 (Pt 2A): 85-101
94. Parsons KC. Protective clothing: heat exchange and physiological objectives. *Ergonomics* 1988; 31: 991-1007
95. Nishi Y, Gagge AP. Direct evaluation of convective heat transfer coefficient by naphthalene sublimation. *J Appl Physiol* 1970; 29: 830-8
96. Kellogg Jr DL, Johnson JM, Kosiba WA. Control of internal temperature threshold for active cutaneous vasodilation by dynamic exercise. *J Appl Physiol* 1991; 71: 2476-82
97. Fortney SM, Nadel ER, Wenger CB, et al. Effect of acute alterations of blood volume on circulatory performance in humans. *J Appl Physiol* 1981; 50: 292-8
98. Mack GW, Nose H, Takamata A, et al. Influence of exercise intensity and plasma volume on active cutaneous vasodilation in humans. *Med Sci Sports Exerc* 1994; 26: 209-16
99. Nadel ER, Fortney SM, Wenger CB. Effect of hydration state on circulatory and thermal regulations. *J Appl Physiol* 1980; 49: 715-21
100. Harrison MH. Effects of thermal stress and exercise on blood volume in humans. *Physiol Rev* 1985; 65: 149-209
101. Dill DB, Costill DL. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol* 1974; 37: 247-8
102. Van Beaumont W. Evaluation of hemoconcentration from hematocrit measurements. *J Appl Physiol* 1972; 31: 712-3
103. Van Beaumont W, Greenleaf JE, Juhos L. Disproportional changes in hematocrit, plasma volume, and proteins during exercise and bed rest. *J Appl Physiol* 1972; 33: 55-61
104. Costill DL, Saltin B. Changes in the ratio of venous to body hematocrit following dehydration. *J Appl Physiol* 1974; 36: 608-10
105. Greenleaf JE, Convertino VA, Mangseth GR. Plasma volume during stress in man: osmolality and red cell volume. *J Appl Physiol* 1979; 47: 1031-8
106. Kolka MA, Stephenson LA, Wilkerson JE. Erythrocyte indices during a competitive marathon. *J Appl Physiol* 1982; 52: 168-72
107. Van Beaumont W. Red cell volume with changes in plasma osmolality during maximal exercise. *J Appl Physiol* 1973; 35: 47-50

108. Shephard RJ. Physiology and biochemistry of exercise. New York: Praeger, 1982.
109. Costill DL, Branam L, Eddy D, et al. Alterations in red cell volume following exercise and dehydration. *J Appl Physiol* 1974; 37: 912-6.
110. Harrison MH, Edwards RJ, Leitch DR. Effect of exercise and thermal stress on plasma volume. *J Appl Physiol* 1975; 39: 925-31.
111. Convertino VA, Kell LC, Greenleaf JE. Plasma volume, renin, and vasopressin responses to graded exercise after training. *J Appl Physiol* 1983; 54: 508-14.
112. Senay LC, Mitchell D, Wyndham CH. Acclimatization in a hot, humid environment: body fluid adjustments. *J Appl Physiol* 1976; 40: 786-96.
113. Shapiro Y, Hubbard RW, Kimbrough CM, et al. Physiological and hematologic responses to summer and winter dry-heat acclimation. *J Appl Physiol* 1981; 50: 792-8.
114. Armstrong LE, Meehan CM. The induction and decay of heat acclimatization in trained athletes. *Sports Med* 1991; 12 (5): 302-12.
115. Fellmann N. Hormonal and plasma volume alterations following endurance exercise: a brief review. *Sports Med* 1992; 13 (1): 37-49.
116. Edwards RJ, Harrison MH. Intravascular volume and protein responses to running exercise. *Med Sci Sports Exerc* 1984; 16: 247-55.
117. Senay Jr LC. Effects of exercise in the heat on body fluid distribution. *Med Sci Sports* 1979; 11: 42-8.
118. Sawka MN, Francesconi RP, Pimental NA, et al. Hydration and vascular fluid shifts during exercise in the heat. *J Appl Physiol* 1984; 56: 91-6.
119. Sawka MN, Young AJ, Francesconi RP, et al. Thermoregulatory and blood responses during exercise at graded hypohydration levels. *J Appl Physiol* 1985; 59: 1394-401.
120. Sawka MN, Toner MM, Francesconi RP, et al. Hypohydration and exercise: effects of heat acclimation, gender, and environment. *J Appl Physiol* 1985; 55: 1147-53.
121. Greenleaf JE, Van Beaumont W, Brock PJ, et al. Plasma volume and electrolyte shifts with heavy exercise in sitting and supine positions. *Am J Physiol* 1979; 236: R206-14.
122. Miles DS, Sawka MN, Olsner RM, et al. Plasma volume shifts during progressive arm and leg exercise. *J Appl Physiol* 1983; 54: 491-5.
123. Mellis B, Eclache JP, Geelen G, et al. Plasma AVP, Neurophysin, renin activity, and aldosterone during submaximal exercise performed until exhaustion in trained and untrained men. *Eur J Appl Physiol* 1980; 44: 141-51.
124. Fortney SM, Nadel ER, Wenger CB, et al. Effect of blood volume on sweating rate and body fluids in exercising humans. *J Appl Physiol* 1981; 51: 1594-600.
125. Senay Jr LC, Pivarski JM. Fluid shifts during exercise. *Exerc Sport Sci Rev* 1983; 13: 333-47.
126. Ladell WSS, Shephard RJ. Aldosterone inhibition and acclimatization to heat. *J Physiol* 1961; 160: 199-209.
127. Kirby CR, Convertino VA. Plasma aldosterone and sweat sodium concentrations after exercise and heat acclimation. *J Appl Physiol* 1986; 61: 967-70.
128. Allan HR, Wilson CG. Influence of acclimatization on sweat sodium concentration. *J Appl Physiol* 1971; 30: 708-12.
129. Harrison MH, Edwards RJ, Craveny MJ, et al. Blood volume and plasma protein responses to heat acclimatization in humans. *J Appl Physiol* 1981; 50: 597-604.
130. Smiles KA, Robinson S. Sodium ion conservation during acclimatization of men to work in the heat. *J Appl Physiol* 1971; 31: 63-9.
131. Costill DL, Costé R, Fink W. Muscle water and electrolytes following varied levels of dehydration in man. *J Appl Physiol* 1976; 40: 6-11.
132. Durkot MJ, Martinez O, Brooks-McQuade D, et al. Simultaneous determination of fluid shifts during thermal stress in a small-animal model. *J Appl Physiol* 1986; 61: 1031-4.
133. Johnson JM. Exercise and the cutaneous circulation. *Exerc Sport Sci Rev* 1992; 20: 59-97.
134. Johnson JM. Nonthermoregulatory control of human skin blood flow. *J Appl Physiol* 1986; 61: 1613-22.
135. Johnson JM, Park MK. Effect of heat stress on cutaneous vascular responses to the initiation of exercise. *J Appl Physiol* 1982; 53: 744-9.
136. Kellogg Jr DL, Johnson JM, Kosiba WA. Competition between cutaneous active vasoconstriction and active vasodilation during exercise in humans. *Am J Physiol* 1991; 261: H1184-9.
137. Toci M, Yamazaki M, Sasaki T, et al. Fall in skin temperature of exercising man. *Br J Sports Med* 1992; 26: 29-32.
138. Johnson JM, Park MK. Effect of upright exercise on threshold for cutaneous vasodilation and sweating. *J Appl Physiol* 1981; 50: 814-8.
139. Kellogg Jr DL, Johnson JM, Kenney WL, et al. Mechanisms of control of skin blood flow during prolonged exercise in humans. *Am J Physiol* 1993; 265: H562-8.
140. Kenney WL, Tankersley CG, Newswanger DL, et al. α_1 -Adrenergic blockade does not alter control of skin blood flow during exercise. *Am J Physiol* 1991; 260: H855-61.
141. Nadel ER, Cafarelli E, Roberts MF, et al. Circulatory regulation during exercise in different ambient temperatures. *J Appl Physiol* 1979; 46: 430-7.
142. Nose H, Mack GW, Shi X, et al. Effect of saline infusion during exercise on thermal and circulatory regulations. *J Appl Physiol* 1990; 69: 609-16.
143. Roberts MF, Wenger CB. Control of skin circulation during exercise and heat stress. *Med Sci Sports* 1979; 11: 36-41.
144. Fortney SM, Wenger CB, Bove JR, et al. Effect of hyperosmolality on control of blood flow and sweating. *J Appl Physiol* 1984; 57: 1688-95.
145. Ohara K. Body temperature and thermal balance [in Japanese]. In: Nakayama T, Iriki M, editors. Handbook of physiological sciences: physiology of energy exchange and thermoregulation. Tokyo: Igaku-shoin, 1987: 89-108.
146. Frascaro P, Schutz Y, Jéquier E. Decreased thermal conductance during the luteal phase of the menstrual cycle in women. *J Appl Physiol* 1990; 69: 2029-33.
147. Holloszy JO. Metabolic consequences of endurance exercise training. In: Horton ES, Terjung RL, editors. Exercise, nutrition, and energy metabolism. New York: Macmillan, 1988: 116-31.
148. Rowell LB. Human cardiovascular control. New York: Oxford University Press, 1993.
149. McArdle WD, Katch FI, Katch VL. Exercise physiology: energy, nutrition, and human performance. 3rd ed. Philadelphia: Lea & Febiger, 1991.

150. Clausen JP. Effect of physical training on cardiovascular adjustments to exercise in man. *Physiol Rev* 1977; 57: 779-815
151. Child JS, Barnard RJ, Taw RL. Cardiac hypertrophy and function in master endurance runners and sprinters. *J Appl Physiol* 1984; 57: 176-81
152. Roy A, Doyon M, Dunneil JG, et al. Endurance vs strength training: comparison of cardiac structures using normal predicted values. *J Appl Physiol* 1988; 64: 2552-7
153. Clausen JP, Trap-Jensen J, Lassen NA. The effects of training on the heart rate during arm and leg exercise. *Scand J Clin Lab Invest* 1970; 26: 295-301
154. McKeazie DC, Fox EL, Cohen K. Specificity of metabolic and circulatory responses to arm or leg interval training. *Eur J Appl Physiol* 1978; 39: 241-8
155. Fortney SM, Wenger CB, Bove JR, et al. Effect of blood volume on forearm venous and cardiac stroke volume during exercise. *J Appl Physiol* 1983; 55: 884-90
156. Saellen JW. Mean body temperature and the control of thermal sweating. *Acta Physiol Pharmacol Neerl* 1966; 14: 99-174
157. Mitchell JW, Nadel ER, Stolwijk JAJ. Respiratory weight losses during exercise. *J Appl Physiol* 1972; 32: 474-6
158. Verde T, Shephard RJ, Corey P, et al. Sweat composition in exercise and in heat. *J Appl Physiol* 1982; 53: 1540-5
159. Moustath JL. Latent heat of vaporization in thermal physiology. *Nature New Biol* 1972; 236: 96
160. Saellen JW, Mitchell D, Wyndham CH. Heat of evaporation of sweat. *J Appl Physiol* 1970; 29: 40-4
161. Wenger CB. Heat of evaporation of sweat: thermodynamic considerations. *J Appl Physiol* 1972; 32: 456-9
162. Mitchell D, Wyndham CH, Atkins AR, et al. Direct measurement of the thermal responses of nude resting men in dry environments. *Pflügers Arch* 1968; 303: 324-43
163. Hardy JD. Heat transfer. In: Newburgh LJJ, editor. *Physiology of heat regulation and the science of clothing*. Philadelphia: Saunders, 1949: 78-108
164. Nielsen B. Regulation of body temperature and heat dissipation at different levels of energy- and heat production in man. *Acta Physiol Scand* 1966; 68: 215-27
165. Shvartz E, Bhattacharya A, Sperinde SJ, et al. Sweating responses during heat acclimation and moderate conditioning. *J Appl Physiol* 1979; 46: 675-80
166. Taylor NAS. Eccrine sweat glands: adaptations to physical training and heat acclimation. *Sports Med* 1986; 3 (6): 387-97
167. Alber-Wallerström B, Holmér I. Efficiency of sweat evaporation in unacclimatized man working in a hot humid environment. *Eur J Appl Physiol* 1985; 54: 480-7
168. Candas V, Libert JP, Vogt JJ. Human skin wettedness and evaporative efficiency of sweating. *J Appl Physiol* 1979; 46: 522-8
169. Candas V, Libert JP, Vogt JJ. Influence of air velocity and heat acclimation on human skin wettedness and sweating efficiency. *J Appl Physiol* 1979; 47: 1194-200
170. Hemmer V, Zeh A, Brück K. Effects of passive heat adaptation and moderate sweatless conditioning on responses to cold and heat. *Eur J Appl Physiol* 1986; 55: 281-9
171. Sato F, Owen M, Matthes R, et al. Functional and morphological changes in the eccrine sweat gland with heat acclimation. *J Appl Physiol* 1990; 69: 232-6
172. Craig FN, Moffitt JT. Efficiency of evaporative cooling from wet clothing. *J Appl Physiol* 1974; 36: 313-6
173. Burton AC. Human calorimetry. II: the average temperature of the tissues of the body. *J Nutr* 1935; 9: 261-80
174. Vallerand AL, Savourey G, Hanniquet A-M, et al. How should body heat storage be determined in humans: by thermometry or calorimetry? *Eur J Appl Physiol* 1992; 65: 286-94
175. Hardy JD, Du Bois EF. Basal metabolism, radiation, convection and vaporization at temperatures of 22 to 35°C. *J Nutr* 1938; 15: 477-97
176. Prim J, Livingstone SD, Reed LD, et al. Body composition and skin temperature variation. *J Appl Physiol* 1990; 68: 540-3
177. Kakitsuba N, Mekjavic IB. Determining the rate of body heat storage by incorporating body composition. *Aviat Space Environ Med* 1987; 58: 301-7
178. Pollock ML, Wilmore JH. *Exercise in health and disease: evaluation and prescription for prevention and rehabilitation*. 2nd ed. Philadelphia: Saunders, 1990
179. Shephard RJ. *Body composition in biological anthropology*. Cambridge: Cambridge University Press, 1991
180. Webb P. Heat storage and body temperature during cooling and rewarming. *Eur J Appl Physiol* 1993; 66: 18-24
181. Vallerand AL, Lüscher R, Schmiegner IF. Computer acquisition and analysis of skin temperature and heat flow data from heat flux transducers. *Comput Methods Programs Biomed* 1989; 30: 279-82
182. Libert J-P, Candas V, Sagot J-C, et al. Contribution of skin thermal sensitivities of large body areas to sweating response. *Jpn J Physiol* 1984; 34: 75-88
183. Nadel ER, Mitchell JW, Stolwijk JAJ. Differential thermal sensitivity in human skin. *Pflügers Arch* 1973; 340: 71-6
184. Mitchell D, Wyndham CH. Comparison of weighting formulas for calculating mean skin temperature. *J Appl Physiol* 1969; 26: 616-22
185. Colin J, Timbal J, Houdas Y, et al. Computation of mean body temperature from rectal and skin temperatures. *J Appl Physiol* 1971; 31: 484-9
186. Hardy JD, Stolwijk JAJ. Partitioned calorimetric studies of man during exposures to thermal transients. *J Appl Physiol* 1966; 21: 1799-806
187. Shvartz E, Gillick Z, Magazanik A. Responses to temperate, cold, and hot environments and the effect of physical training. *Aviat Space Environ Med* 1977; 48: 254-60
188. Bittel JHM. Heat debt as an index for cold adaptation in men. *J Appl Physiol* 1987; 62: 1627-34
189. Savourey G, Vallerand AL, Bittel JHM. General and local cold adaptation after a ski journey in a severe arctic environment. *Eur J Appl Physiol* 1992; 64: 99-105
190. Vallerand AL, Savourey G, Bittel JHM. Determination of heat debt in the cold: partitioned calorimetry vs conventional methods. *J Appl Physiol* 1992; 72: 1380-5
191. Nielsen B. Thermoregulation in rest and exercise. *Acta Physiol Scand* 1969; Suppl. 323: 1-74
192. Stolwijk JAJ, Nadel ER. Thermoregulation during positive and negative work exercise. *Fed Proc* 1973; 32: 1607-13
193. Stolwijk JAJ, Hardy JD. Partitioned calorimetric studies of responses of man to thermal transients. *J Appl Physiol* 1966; 21: 967-77
194. Holmér I, Eklund S. Physiological evaluation of the resistance to evaporative heat transfer by clothing. *Ergonomics* 1981; 24: 63-74

195. Valerand AL, Michas RD, Frim J, et al. Heat balance of subjects wearing protective clothing with a liquid- or air-cooled vest. *Aviat Space Environ Med* 1991; 62: 383-91
196. McLellan TM. Work performance at 40°C with Canadian Forces biological and chemical protective clothing. *Aviat Space Environ Med* 1993; 64: 1094-100
197. Louhevaara VA. Physiological effects associated with the use of respiratory protective devices: a review. *Scand J Work Environ Health* 1984; 10: 275-81
198. Raven PB, Dodson AT, Davis TO. The physiological consequences of wearing industrial respirators: a review. *Am Ind Hyg Assoc J* 1979; 40: 517-34
199. McLellan TM, Frim J. Heat strain in the Canadian Forces chemical defence clothing: problems and solutions. *Can J Appl Physiol* 1994; 19: 379-99
200. Goldman RP. Standards for human exposure to heat. In: Matijevic TB, Banister EW, Morrison JB, editors. *Environmental ergonomics: sustaining human performance in harsh environments*. Philadelphia: Taylor & Francis, 1988: 99-136
201. Armstrong LE, Szlyk PC, Sils IV, et al. Prediction of the exercise-heat tolerance of soldiers wearing protective overgarments. *Aviat Space Environ Med* 1991; 62: 673-7
202. Pandolf KB, Burt RL, Goldman RP. Role of physical fitness in heat acclimatization, decay and reinduction. *Ergonomics* 1977; 20: 399-408
203. Coggan AR, Coyle EF. Carbohydrate ingestion during prolonged exercise: effects on metabolism and performance. *Exerc Sport Sci Rev* 1991; 19: 1-40
204. Coyle EF, Coggan AR. Effectiveness of carbohydrate feeding in delaying fatigue during prolonged exercise. *Sports Med* 1984; 1 (6): 446-58
205. Hubbard RW, Armstrong LE. The heat illnesses: biochemical, ultrastructural, and fluid-electrolyte considerations. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. *Human performance physiology and environmental medicine at terrestrial extremes*. Indianapolis: Benchmark, 1988: 305-59
206. Martin BJ. Sleep deprivation and exercise. In: Pandolf KB, editor. *Exercise and sport sciences reviews*. New York: Macmillan, 1986: 213-29
207. Maughan RJ, Noakes TD. Fluid replacement and exercise stress: a brief review of studies on fluid replacement and some guidelines for the athlete. *Sports Med* 1991; 12 (1): 16-31
208. Noakes TD. Fluid replacement during exercise. In: Holloszy JO, editor. *Exercise and sport sciences reviews*. Baltimore: Williams & Wilkins, 1993: 297-330
209. Sawka MN, Young AJ. Acute polycythemia and human performance during exercise and exposure to extreme environments. In: Pandolf KB, editor. *Exercise and sport sciences reviews*. Baltimore: Williams & Wilkins, 1989: 265-93
210. Shephard RJ. Sleep, biorhythms and human performance. *Sports Med* 1984; 1 (1): 11-37
211. Stephenson LA, Kolka MA. Effect of gender, circadian period and sleep loss on thermal responses during exercise. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. *Human performance physiology and environmental medicine at terrestrial extremes*. Indianapolis: Benchmark Press, 1988: 267-304
212. Stephenson LA, Kolka MA. Thermoregulation in women. In: Holloszy JO, editor. *Exercise and sport sciences reviews*. Baltimore: Williams & Wilkins, 1993: 231-62
213. Armstrong LE, Costill DL, Fink WJ. Influence of diuretic-induced dehydration on competitive running performance. *Med Sci Sports Exerc* 1985; 17: 456-61
214. Caldwell JE, Ahonen E, Nousiainen U. Differential effects of sauna-, diuretic-, and exercise-induced hypohydration. *J Appl Physiol* 1984; 57: 1018-23
215. Greenleaf JE, Castle BL. Exercise temperature regulation in man during hypohydration and hyperhydration. *J Appl Physiol* 1971; 30: 847-53
216. Maughan RJ, Fenn CB, Gleason M, et al. Metabolic and circulatory responses to the ingestion of glucose polymer and glucose/electrolyte solutions during exercise in man. *Eur J Appl Physiol* 1987; 56: 356-62
217. Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol* 1992; 73: 1340-50
218. Sawka MN, Knowlton RG, Critz JB. Thermal and circulatory responses to repeated bouts of prolonged running. *Med Sci Sports* 1979; 11: 177-80
219. Sawka MN, Knowlton RG, Glaser RM, et al. Effect of prolonged running on physiological responses to subsequent exercise. *J Hum Ergol* 1979; 8: 83-90
220. Sawka MN, Francesconi RP, Young AJ, et al. Influence of hydration level and body fluids on exercise performance in the heat. *JAMA* 1984; 252: 1163-9
221. Candas V, Libert JP, Brandenberger G, et al. Hydration during exercise: effects on thermal and cardiovascular adjustments. *Eur J Appl Physiol* 1986; 55: 113-22
222. Senay Jr LC. Temperature regulation and hypohydration: a singular view. *J Appl Physiol* 1979; 47: 1-7
223. Silva NL, Boulant JA. Effects of osmotic pressure, glucose, and temperature on neurons in preoptic tissue slices. *Am J Physiol* 1984; 247: R335-45
224. Nielsen B, Hansen G, Jorgensen SO, et al. Thermoregulation in exercising man during dehydration and hyperhydration with water and saline. *Int J Biometeor* 1971; 15: 195-200
225. Roberts MF, Wenger CB. Control of skin blood flow during exercise by thermal reflexes and baroreflexes. *J Appl Physiol* 1980; 48: 717-23
226. Bar-Or O, Dotan R, Inbar O, et al. Voluntary hypohydration in 10- to 12-year-old boys. *J Appl Physiol* 1980; 48: 104-8
227. Engell DB, Maller O, Sawka MN, et al. Thirst and fluid intake following graded hypohydration levels in humans. *Physiol Behav* 1987; 40: 229-36
228. Nee H, Mack GW, Shi X, et al. Role of osmolality and plasma volume during rehydration in humans. *J Appl Physiol* 1988; 65: 325-31
229. Rolls BJ, Wood RJ, Rolls ET, et al. Thirst following water deprivation in humans. *Am J Physiol* 1980; 239: R476-82
230. Kavanagh T, Shephard RJ. On the choice of fluid for the hydration of middle-aged marathon runners. *Br J Sports Med* 1977; 11: 26-35
231. Barr SI, Costill DL, Fink WJ. Fluid replacement during prolonged exercise: effects of water, saline, or no fluid. *Med Sci Sports Exerc* 1991; 23: 811-7
232. Hamilton MT, Gonzalez-Alonso J, Montain SJ, et al. Fluid replacement and glucose infusion during exercise prevent cardiovascular drift. *J Appl Physiol* 1991; 71: 871-7

233. Montain SJ, Coyle EF. Fluid ingestion during exercise increases skin blood flow independent of increases in blood volume. *J Appl Physiol* 1992; 73: 903-10
234. Maughan RJ, Penn CE, Leiper JB. Effects of fluid, electrolyte and substrate ingestion on endurance capacity. *Eur J Appl Physiol* 1989; 58: 481-6
235. Gisolfi CV, Copping JR. Thermal effects of prolonged treadmill exercise in the heat. *Med Sci Sports* 1974; 6: 108-13
236. Costill DL, Saltin B. Factors limiting gastric emptying during rest and exercise. *J Appl Physiol* 1974; 37: 679-83
237. McArthur KE, Feldman M. Gastric acid secretion, gastrin release, and gastric emptying in humans as affected by liquid meal temperature. *Am J Clin Nutr* 1989; 49: 51-4
238. Sun WM, Houghton LA, Read NW, et al. Effect of meal temperature on gastric emptying of liquids in man. *Gut* 1988; 29: 302-5
239. Mitchell JB, Costill DL, Houmard JA, et al. Gastric emptying: influence of prolonged exercise and carbohydrate concentration. *Med Sci Sports Exerc* 1989; 21: 269-74
240. Owen MD, Kregel KC, Wall PT, et al. Effects of ingesting carbohydrate beverages during exercise in the heat. *Med Sci Sports Exerc* 1986; 18: 568-73
241. Rehrer NJ, Beckers E, Brouns F, et al. Exercise and training effects on gastric emptying of carbohydrate beverages. *Med Sci Sports Exerc* 1989; 21: 540-9
242. Buick FJ, Gledhill N, Proese AB, et al. Effect of induced erythrocythemia on aerobic work capacity. *J Appl Physiol* 1980; 48: 636-42
243. Kanstrup I-L, Ekblom B. Blood volume and hemoglobin concentration as determinants of maximal aerobic power. *Med Sci Sports Exerc* 1984; 16: 256-62
244. Muza SR, Sawka MN, Young AJ, et al. Elite special forces: physiological description and ergogenic influence of blood reinfusion. *Aviat Space Environ Med* 1987; 58: 1001-4
245. Robertson RJ, Glicker R, Metz KF, et al. Hemoglobin concentration and aerobic work capacity in women following induced erythrocythemia. *J Appl Physiol* 1984; 57: 568-75
246. Francesconi RP, Sawka MN, Dennis RC, et al. Autologous red blood cell reinfusion: effects on stress and fluid regulatory hormones during exercise in the heat. *Aviat Space Environ Med* 1988; 59: 133-7
247. Sawka MN, Gonzalez RR, Young AJ, et al. Polycythemia and hydration: effects on thermoregulation and blood volume during exercise-heat stress. *Am J Physiol* 1988; 255: R456-63
248. Gisolfi CV, Summers RW, Schedl HP, et al. Human intestinal water absorption: direct vs indirect measurements. *Am J Physiol* 1990; 258: G216-22
249. Gisolfi CV, Spranger KJ, Summers RW, et al. Effects of cycle exercise on intestinal absorption in humans. *J Appl Physiol* 1991; 71: 2518-27
250. Coyle EF, Montain SJ. Benefits of fluid replacement with carbohydrate during exercise. *Med Sci Sports Exerc* 1992; 24: S324-30
251. Williams C, Nute MG, Broadbank L, et al. Influence of fluid intake on endurance running performance: a comparison between water, glucose and fructose solutions. *Eur J Appl Physiol* 1990; 60: 112-9
252. Seidman DS, Ashkenazi I, Arnon R, et al. The effects of glucose polymer beverage ingestion during prolonged outdoor exercise in the heat. *Med Sci Sports Exerc* 1991; 23: 458-62
253. Seiple RS, Vivian VM, Fox EL, et al. Gastric-emptying characteristics of two glucose polymer-electrolyte solutions. *Med Sci Sports Exerc* 1983; 15: 366-9
254. Sole CC, Noakes TD. Faster gastric emptying for glucose-polymer and fructose solutions than for glucose in humans. *Eur J Appl Physiol* 1989; 58: 605-12
255. Yaspelkis BB III, Ivy JL. Effect of carbohydrate supplements and water on exercise metabolism in the heat. *J Appl Physiol* 1991; 71: 680-7
256. Konikoff F, Shoenfeld Y, Magazanik A, et al. Effects of salt loading during exercise in a hot dry climate. *Blomed Pharmacother* 1986; 40: 296-300
257. Costill DL. Sweating: its composition and effects on body fluids. In: Milvy P, editor. *The marathon: physiological, medical, epidemiological, and psychological studies*. New York: The New York Academy of Sciences, 1977: 160-74
258. Costill DL, Branam G, Fink W, et al. Exercise induced sodium conservation: changes in plasma renin and aldosterone. *Med Sci Sports* 1976; 8: 209-13
259. Irving RA, Noakes TD, Irving GA, et al. The immediate and delayed effects of marathon running on renal function. *J Urol* 1986; 136: 1176-80
260. Irving RA, Noakes TD, Burger SC, et al. Plasma volume and renal function during and after ultramarathon running. *Med Sci Sports Exerc* 1990; 22: 581-7
261. Irving RA, Noakes TD, Raine RL, et al. Transient oliguria with renal tubular dysfunction after a 90km running race. *Med Sci Sports Exerc* 1990; 22: 756-61
262. Armstrong LE, Hubbard RW, Szlyk PC, et al. Voluntary dehydration and electrolyte losses during prolonged exercise in the heat. *Aviat Space Environ Med* 1985; 56: 765-70
263. Dill DB, Sjolvist LF, McLean, et al. Capacity of young males and females for running in desert heat. *Med Sci Sports* 1977; 9: 137-42
264. Francis KT, MacGregor R III. Effect of exercise in the heat on plasma renin and aldosterone with either water or a potassium-rich electrolyte solution. *Aviat Space Environ Med* 1978; 49: 461-5
265. Armstrong LE, Costill DL, Fink WJ, et al. Effects of dietary sodium on body and muscle potassium content during heat acclimation. *Eur J Appl Physiol* 1985; 54: 391-7
266. Armstrong LE, Costill DL, Fink WJ. Changes in body water and electrolytes during heat acclimation: effects of dietary sodium. *Aviat Space Environ Med* 1987; 58: 143-8
267. Knochel JP, Vertel RM. Salt loading as a possible factor in the production of potassium depletion, rhabdomyolysis, and heat injury. *Lancet* 1967; 1: 659-61
268. Nose H, Morita M, Yawata T, et al. Recovery of blood volume and osmolality after thermal dehydration in rats. *Am J Physiol* 1986; 251: R492-8
269. Wade CE. Response, regulation, and actions of vasopressin during exercise: a review. *Med Sci Sports Exerc* 1984; 16: 506-11
270. Brandenberger G, Candau V, Follenius M, et al. The influence of the initial state of hydration on endocrine responses to exercise in the heat. *Eur J Appl Physiol* 1989; 58: 674-9
271. Thrasher TN, Nistal-Herrera JF, Keil LC, et al. Satiety and inhibition of vasopressin secretion after drinking in dehydrated dogs. *Am J Physiol* 1981; 240: E394-401

272. Thrasher TN, Wade CE, Keil LC, et al. Sodium balance and aldosterone during dehydration and rehydration in the dog. *Am J Physiol* 1984; 247: R76-83
273. Hessemer V, Brück K. Influence of menstrual cycle on thermoregulatory, metabolic, and heart rate responses to exercise at night. *J Appl Physiol* 1985; 59: 1911-7
274. Pivarnik JM, Marichal CJ, Spillman T, et al. Menstrual cycle phase affects temperature regulation during endurance exercise. *J Appl Physiol* 1992; 72: 543-8
275. Silva NL, Boulant JA. Effects of testosterone, estradiol, and temperature on neurons in preoptic tissue slices. *Am J Physiol* 1986; 250: R625-32
276. Cannon JO, Dinarello CA. Increased plasma interleukin-1 activity in women after ovulation. *Science* 1985; 227: 1247-9
277. Hessemer V, Brück K. Influence of menstrual cycle on shivering, skin blood flow, and sweating responses measured at night. *J Appl Physiol* 1985; 59: 1902-10
278. Stephenson LA, Kolka MA. Menstrual cycle phase and time of day alter reference signal controlling arm blood flow and sweating. *Am J Physiol* 1985; 249: R186-91
279. Avellini BA, Kamei E, Krajewski JT. Physiological responses of physically fit men and women to acclimation to humid heat. *J Appl Physiol* 1980; 49: 254-61
280. Frye AJ, Kamon E. Responses to dry heat of men and women with similar aerobic capacities. *J Appl Physiol* 1981; 50: 65-70
281. Frye AJ, Kamon E. Sweating efficiency in acclimated men and women exercising in humid and dry heat. *J Appl Physiol* 1983; 54: 972-7
282. Frye AJ, Kamon E, Webb M. Responses of menstrual women, amenorrheal women, and men to exercise in a hot, dry environment. *Eur J Appl Physiol* 1982; 48: 279-88
283. Carpenter AJ, Nunealey SA. Endogenous hormones subtly alter women's response to heat stress. *J Appl Physiol* 1988; 65: 2313-7
284. Little MA, Rummel JA. Circadian variations in thermal and metabolic responses to heat exposure. *J Appl Physiol* 1971; 31: 556-61
285. Reilly T, Brooks GA. Investigation of circadian rhythms in metabolic responses to exercise. *Ergonomics* 1982; 25: 1093-107
286. Minors DS, Waterhouse JM. Endogenous and exogenous components of circadian rhythms when living on a 21-hour day. *Int J Chronobiol* 1981; 8: 31-48
287. Wever RA. Fractional desynchronization of human circadian rhythms: a method for evaluating entrainment limits and functional interdependencies. *Pflügers Arch* 1983; 396: 128-37
288. Stephenson LA, Wenger CB, O'Donovan BH, et al. Circadian rhythm in sweating and cutaneous blood flow. *Am J Physiol* 1984; 246: R321-4
289. Wenger CB, Roberts MF, Stolkwijk JAJ, et al. Nocturnal lowering of thresholds for sweating and vasodilation. *J Appl Physiol* 1976; 41: 15-9
290. Henane R, Bugnot A, Roussel B, et al. Variations in evaporation and body temperatures during sleep in man. *J Appl Physiol* 1977; 42: 50-5
291. Sagot JC, Amorós C, Candas V, et al. Sweating responses and body temperatures during nocturnal sleep in humans. *Am J Physiol* 1987; 252: R462-70
292. Kolka MA, Stephenson LA. Exercise thermoregulation after prolonged wakefulness. *J Appl Physiol* 1988; 64: 1575-9
293. Kolka MA, Martin BJ, Elizondo RS. Exercise in a cold environment after sleep deprivation. *Eur J Appl Physiol* 1984; 53: 282-5
294. Sawka MN, Gonzalez RR, Pandolf KB. Effects of sleep deprivation on thermoregulation during exercise. *Am J Physiol* 1984; 246: R72-7
295. Pyley MJ, Shephard RJ, Davis GM, et al. Sleep deprivation and cardiorespiratory function: influence of intermittent submaximal exercise. *Eur J Appl Physiol* 1987; 56: 338-44
296. Buono MJ, Sjöholm NT. Effect of physical training on peripheral sweat production. *J Appl Physiol* 1988; 65: 811-4
297. Shapiro Y, Pandolf KB, Goldman RF. Sex differences in acclimation to a hot-dry environment. *Ergonomics* 1980; 23: 635-42
298. Shapiro Y, Pandolf KB, Avellini BA, et al. Heat balance and transfer in men and women exercising in hot-dry and hot-wet conditions. *Ergonomics* 1981; 24: 375-86
299. Anderson RK, Kenney WL. Effect of age on heat-activated sweat gland density and flow during exercise in dry heat. *J Appl Physiol* 1987; 63: 1089-94
300. Kenney WL, Anderson RK. Responses of older and younger women to exercise in dry and humid heat without fluid replacement. *Med Sci Sports Exerc* 1988; 20: 155-60
301. Kenney WL, Tankersley CG, Newswanger DL, et al. Age and hypohydration independently influence the peripheral vascular response to heat stress. *J Appl Physiol* 1990; 68: 1902-8
302. Kolka MA, Stephenson LA, Rock PB, et al. Local sweating and cutaneous blood flow during exercise in hypobaric environments. *J Appl Physiol* 1987; 62: 2224-9
303. Kolka MA, Stephenson LA, Gonzalez RR. Depressed sweating during exercise at altitude. *J Therm Biol* 1989; 14: 167-70
304. Havenith G, Van Middendorp H. The relative influence of physical fitness, acclimatization state, anthropometric measures and gender on individual reactions to heat stress. *Eur J Appl Physiol* 1990; 61: 419-27
305. Horstman DH, Christensen E. Acclimatization to dry heat: active men vs active women. *J Appl Physiol* 1982; 52: 825-31
306. Shapiro Y, Pandolf KB, Avellini BA, et al. Physiological responses of men and women to humid and dry heat. *J Appl Physiol* 1980; 49: 1-8
307. Smolander J, Korhonen O, Ilmarinen R. Responses of young and older men during prolonged exercise in dry and humid heat. *Eur J Appl Physiol* 1990; 61: 413-8
308. Tankersley CG, Smolander J, Kenney WL, et al. Sweating and skin blood flow during exercise: effects of age and maximal oxygen uptake. *J Appl Physiol* 1991; 71: 236-42
309. Delamarche P, Bittel J, Lacour JR, et al. Thermoregulation at rest and during exercise in prepubertal boys. *Eur J Appl Physiol* 1990; 60: 436-40
310. Dawson B. Exercise training in sweat clothing in cool conditions to improve heat tolerance. *Sports Med* 1994; 17 (4): 233-44
311. Ihzuka H, Hori S, Akamatsu T. Seasonal variations of physiological responses to heat of subtropical and temperate natives. *Int J Biometeor* 1986; 30: 107-13

Correspondence and reprints: Prof. Roy J. Shephard, School of Physical and Health Education, University of Toronto, 320 Huron Street, Toronto, Ontario M5S 1A1, Canada.